THE TAMILNADU Dr. M.G.R. MEDICAL UNIVERSITY

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“SPIROMETRY IN ASYMPTOMATIC SMOKERS”

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BRANCH I

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EXAMINATION

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CERTIFICATE

This is to certify that this dissertation entitled **"SPIROMETRY IN ASYMPTOMATIC SMOKERS"** is the bonafide record work done by **Dr. I.LAKSHMI,** submitted as partial fulfillment for the requirements of M.D. Degree Examinations, General Medicine (Branch I) to be held in APRIL 2013.

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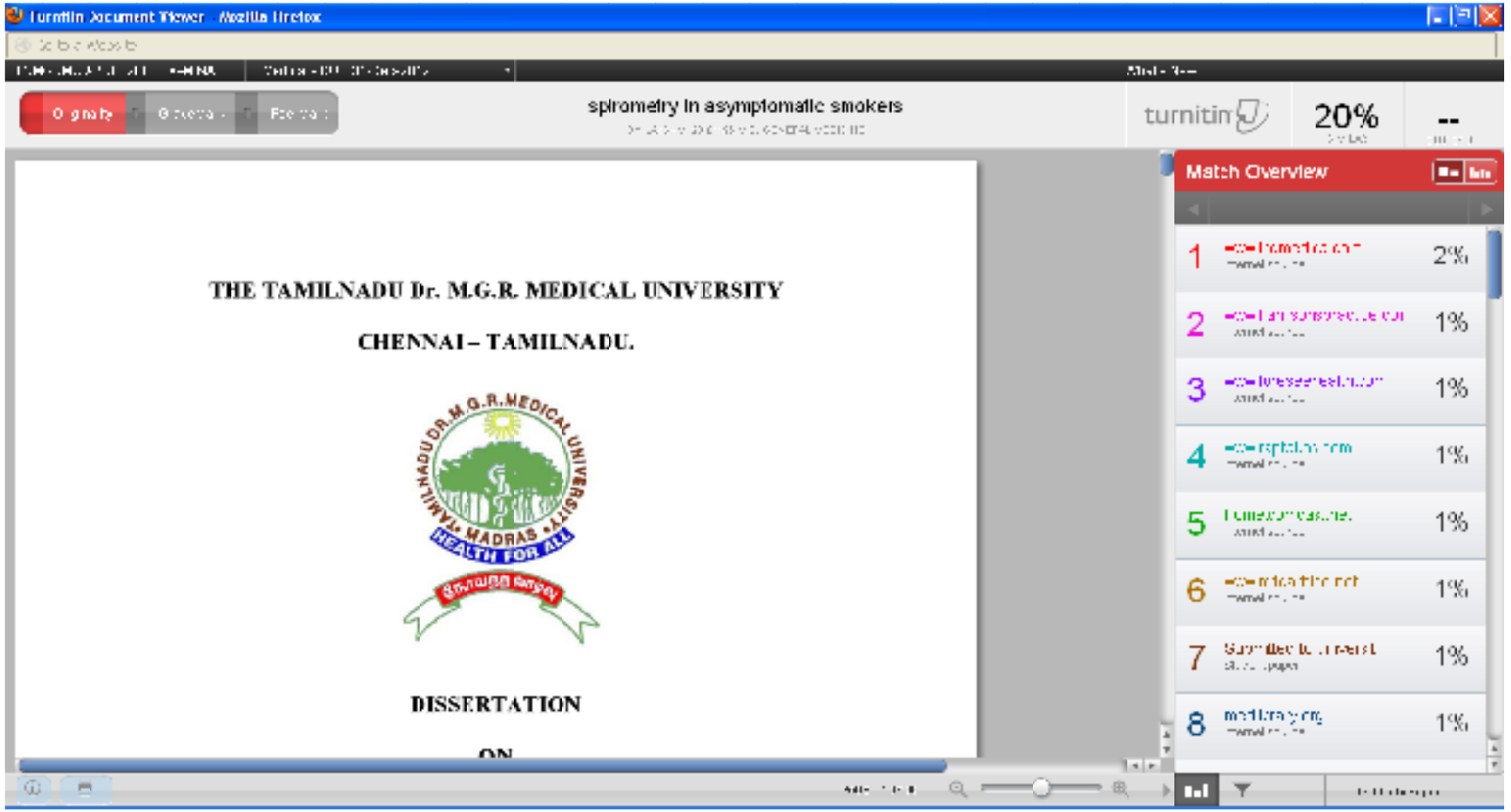
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CONTENTS

ABBREVIATIONS

FVC	-	Forced vital capacity
VC	-	Vital capacity
FEV1	-	Forced expiratory volume in 1s
FEV1/FVC	-	Ratio of FEV1 to FVC
FEF25-75%	-	Forced expiratory flow rate between 25 and 75% of the VC
TLC	-	Total lung capacity
RV	-	Residual volume
MMFR	-	Maximal midexpiratory flow rate
MIP	-	Maximal inspiratory pressure
MEP	-	Maximal expiratory pressure

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is at present, the sixth leading cause of mortality and twelfth leading cause of morbidity worldwide. Smoking is one of the most important major risk factor leading to the development of COPD. The risk of morbidity and mortality increases with both the quantum of smoking and duration in years.

Therefore the incidence of COPD can be prevented of if we do screening of the smokers without any respiratoy symptoms in their early age itself. This can be achieved by doing spirometry in smokers. Spirometry measures the pulmonary function by measuring the air flow rates, lung volumes in the form of measuring FEV1, FVC, PEF 25 – 75% FEV1/ FVC. It should be compared to their normal predicted values.

If there is a decline in any of the above values, then the risk of occurrence of COPD in the near future can be assessed. Patient can be advised to quit smoking, as cessation of smoking can halt the incidence and progression of the disease.

AIM OF STUDY

1. To evaluate the pulmonary function test parameters in asymptomatic smokers.
2. To compare the spirometric findings in asymptomatic smokers to their expected values.
3. To identify the asymptomatic COPD among smokers, so that cessation of smoking would halt their progression.
4. To identify the degrees of deterioration in PFT.
5. To correlate the dysfunction with the quantum of smoking.

REVIEW OF LITERATURE

Anatomy of the lungs:[35]

The respiratory system is made up of units for gas exchange (ie) the lungs and a pump which is necessary to ventilate the lungs. The pump is made up of the wall of the chest, the muscles of respiration which can either increase or decrease the size of thoracic carity, the brain which has the central control over respiration.

At the rest, man breathes normally 12-15 times per minute. Every time about 500ml of air enters the lung lion 6 lt/min of air is inspired or expired. So 250ml of O_2 enters the body and 200ml of Co_2 is excreted from the body per minute.

Air Passages :

The air passes through the nose, pharynx where it is warmed, then it pass down through trachea and bronchioles, respiratory bronchiolos, alveolar ducts and to the alveoli. It is the alveoli, where gas exchange ocurs. The first 16 generations are mainly concerned with the transport of gas from and to the exterior. They are constituted by bronchi, bronchioles and terminal bronchioles.

The remaining seven generations which forms the transititional and respiratory passages are concerned with gas exchange. They are constituted by respiratory bronchioles, alveolar ducts and alveoli. The

cross sectional area is increased form 2.5cm_2 in trachea to 11,800 cm_2 in alveoli due to these multiple divisions. So, the velocity of the flow of the air is very much declined.

Blood supply to the lungs :

All most, all the blood pass through the pulmonary artery which reaches the capillary bed. Here blood is oxygenated. Then it returns to the left atrium of the heart through the pulmonary veins. Bronchial arteries from systemic circulation also supplies the lungs.

Mechanics of respiration :[34]

The lungs and chest wall are elastic structures. Inspiration is an active process expiration is a passive process.

Respiratory volumes and Capacities :

1) Tidal Volume :

It is the amount of air which is inhaled or exhaled in one breath, while the person being relaxed and breathing quietly, normally, it is about 500ml.

2) Inspiratory Reserve Volume (IRV) :

Amount of air which can be inspired in excess of that tidal volume with a maximal inspiratory effort. It is about 3000ml.

3) Expiratory Reserve Volume (ERV) :

It is the amount of air which is expired in excess to that of tidal expiration which can be exhaled with a maximal expiratory effort. It is about 1200ml.

4) Residual Volume (RV):

It is the amount of air which remains in the lungs after a maximum exhalation. It is about 1200ml.

5) Vital Capacity (VC) :

It is the amount of air which can be expired with a maximum effort after a maximum inspiration. (ie) ERV + TV + IRV. It is about 4700ml.

6) Inspiratory Capacity (IC) :

It is the maximum amount of air that can be inspired after a normal expiration. It is about 3500ml.

7) Functional Residual Capacity :

It is the amount of air that remain in the lungs after a normal expiration. It is about 2400ml.

8) Total Lung Capacity :

It is the maximum amount of air which the lung can accomodate. It constitutes about 5900ml.

SPIROMETRY

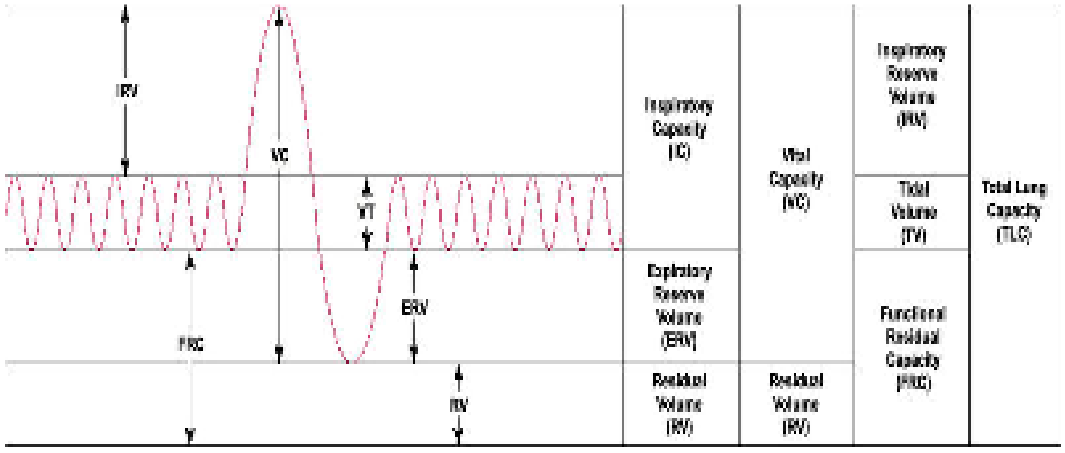
Procedure

The test is performed in the sitting position and airflow is recorded as forced and sustained expiration followed by inspiration.3 efforts which have less than 5% variability between each other are selected and the best effort is used for interpretation.

Spirometry Measurements:[4]

FVC-Total volume of air exhaled with a maximal effort after deep inspiration.

FEV1-Volume of air expired in the first second after deep inspiration.



Other parameters are FEF25-75%,which is the midportion of expiration and vital capacity.

Spirometry

Spirometry is the measurement of airflow during inspiration and expiration. It is used to evaluate the pulmonary function in people with obstructive or restrictive lung diseases such as COPD, bronchial asthma, interstitial lung diseases etc.

Indications:

- To confirm the presence or absence of respiratory disease in a person with signs or symptoms or presence of abnormal chest x – ray or ABG.
- To quantify the degree of known lung diseases.
- To follow up the change in severity of the lung functions.
- To assess the response to environmental and occupational response.
- To assess the risk of surgical procedures which can affect the lungs.
- To differentiate between obstructive and restrictive pattern of lung diseases.

Contra indications:

- Haemoptysis of unknown origin.
- Pnemothorax.
- Unstable cardiac status/ recent myocardial infarction.
- Pulmonary embolism.
- Thoracic,cerebral and abdominal aneurysm.
- Recent cataract surgery.
- Presence of any acute disease conditions.
- Recent surgery done in abdomen or thorax.

Complications:

- Pnemothorax.
- Raised intracranial tension.
- Syncope,light headedness,dizziness.
- Chest pain.
- Paroxysmal cough.
- Nosocomial infections.
- Bronchospasm.

Normal Spirometric Values

Parameters	Predicted Normal Values	% Predicted Baseline
FVC (lt)	5	70%
FEV_1 (lt)	4	50%
FEV_1/FVC(lt/Sec)	80	--
FEF 25-75%	4	50
PEFR (lt / Sec)	8	75

PATTERNS INTERPRETED BY SPIROMETRY

TYPES	FEV_1	FVC	FEV1/FVC
Obstructive	↓↓	↓/N	↓
Restrictive	↓	↓↓	↑/N

Smoking[16]

Smoking is the practice of burning tobacco and inhaling the resulting smoke (consisting of particle and gaseous phases). It causesdamage to cells on the human body, no matter in which form it is packaged. However, it is said some forms of smoking tobacco are less harmful than others.

Smoking in India :

- 30% of the population are 15 years or older in age group.
- 47% men and 14% of women—either smoke or chew tobacco.
- Tobacco consumption was significantly higher in lower socioeconomic population.

Types of Smoking Tobacco:

I. Cigarettes:

- Manufactured cigarettes are the most popular forms of smoking tobacco worldwide.
- They are made by machine and put together using shredded or re-used tobacco along with lots of different chemicals.
- They also contain nicotine, which can make them very addictive.

II. Cigars:

- Cigars are made up of tobacco that has been air-cured and fermented. The filling is held together in a wrapper that also made from tobacco.
- Cigars can come in a variety of sizes, though generally they are bigger than cigarettes. They were previously thought of as an item smoked by wealthier, older men.
- Trends have changed in recent years with much younger people, including women.
- They are said to be a safer alternative to cigarettes because the user should not inhale when smoking a cigar, meaning no smoke reaches the lungs.
- However, they are still a danger to a person's health and can still cause cancer in and around the mouth

III. Pipe :[5]

- Pipe tobacco is normally a blend of tobaccos that may also contain different kinds of additives.
- Pipes are made from a variety of different materials. The user places a flame directly onto the tobacco and smokes the fumes through a hole.

- They are also thought to be a safer alternative to cigarettes because the smoke does not have to be inhaled by the user.
- However, cancer of the lip is more prevalent in pipe smokers than any other form of tobacco smoking.

Bidis :

- Bidis are the most popular form of smoking tobacco in India.
- They have a only a small amount of tobacco, which is wrapped in dried temburni leaves.
- They can come in either flavoured or unflavoured form and are imported into the United States from South Asia.
- Bidis are said to have a higher concentration of tar and nicotine than manufactured cigarettes
- The user usually has to smoke it harder to keep it alight.

Chemicals associated with smoking: [30]

- The chemicals in cigarettes and tobacco smoke make smoking harmful.
- Tobacco smoke contains over 4,000 different chemicals, at least 50 are known to be carcinogens (cause cancer in humans) and many are poisonous.

CHEMICALS IN CIGARETTE

Benzene (petrol additive)

- A colourless agent which is a cyclic hydrocarbon and it is obtained from coal and petroleum.It is used as a solvent for fuels and chemicals which is present in cigarette smoker also.
- It is a well known carcinogen which is associated with the development of leukaemia.

Formaldehyde (embalming fluid)

- A colourless liquid, which is highly poisonous,and it is used to preserve dead bodies by embalmation. – which is present in cigarette smoke.

- Known to cause carcinoma, respiratory, dermatological and gastrointestinal problems.

Ammonia (Toilet Cleaner)

- Used as a flavouring agent,can liberate nicotine from tobacco and turn it into a gas.
- Most frequently seen in dry cleaning fluids.

Acetone (Nail Polish Remover)

- It is a fragrant volatile liquid which is a ketone, and used as a solvent, for example, to remove nail polish.
- Present in tobacco smoke.

Tar

- Particulate matter which can be drawn into lungs during inhalation from a lighted cigarette. After inhalalation,in the lung of smokers, the tar condenses more than 75 per cent and gets deposited in the respiratory tract .

Nicotine (Insecticide/ Addictive Drug)

- One of the most common addictive substances commonly amneble to man,which is a powerful and fast-acting poison.
- This is the chemical commonly causing addiction in humans.

Carbon Monoxide (car exhaust fumes)

- An odourless, tasteless gas which is a poisonous gas,and is rapidly fatal when inhaled in huge amounts.
- The same gas is also produced in car exhausts
- The main gas in tobacco smoke, formed when the cigarette is lighted.

Diseases associated with smoking: [16]

- Smokers frequently suffer from respiratory infections
- Smoking affects almost all organs of the body. Smoking causes several diseases and detoriates the health of smokers .

Smoking and Increased Health Risks[32]

Compared with nonsmokers, smoking is estimated to increase the risk of

- Increase in incidence of coronary heart disease by two to four times,
- Increase in incidence of cerebro vascular accidents by two to four times,
- Males developing bronchogenic carcinoma by twenty three times,
- Females developing bronchogenic carcinoma by thirteen times, and Death due to chronic obstructive lung diseases (such as chronic bronchitis and emphysema) by twelveto thirteen times.[1]

Smoking and Diseases related to heart:

- Cigarette smoking leads to reduction of circulation by occlusion of the arteries and leads to increased risk of development of peripheral arterial disease like gangrene of the extremities that can lead to symptoms like pain .
- Smoking can lead to the development of aneurysm of the abdominal aorta (i.e., thinning of the arterial walls of the important vessel of the body - the aorta in its course through the abdomen).

Smoking and Respiratory Disease

- Smoking causes bronchogenic carcinoma.
- Smoking causes respiratory diseases (e.g., emphysema, bronchitis, chronic airway obstruction) by destruction of the airways and alveoli (i.e., small air sacs) of the lungs.

Smoking and Cancer :[31]

Smoking causes the following cancers:

- Leukemias like AML.
- Carcinoma of urinary bladder.
- Cervical carcinoma.
- Esophageal carcinoma.
- Renal cell carcinoma.
- Laryngeal carcinoma.

- Bronchogenic carcinoma.
- Cancer of the cheek,tongue,lips.
- Pancreatic carcinoma.
- Pharyngeal carcinoma.
- Stomach carcinoma.
- Hepatocellular carcinoma

Smoking and Other Health Hazards:

Smoking has several deleterious effects during pregnancy and early childhood period, including increased risk for

- Infertility,
- Premature labour
- Intrauterine growth retardation,
- Stillbirth,
- Birth weight lower than normal, and
- Sudden infant death syndrome (SIDS).

Smoking is associated with the following deleterious health effects:

- Postmenopausal women with h/o smoking develop osteoporosis more frequently than women who had never smoked.
- Women with h/o smoking will have an increased risk for fracture of hip joint.

Other Disease Associated with Smoking

Acute coronary syndrome	Bronchial asthma
Coronary heart disease	Diabetes mellitus
Cardiovascular disease	Peptic ulcers
Congestive cardiac failure	Cataracts
Cerebrovascular disease	Gum disease
Atherosclerosis	Systemic hypertension
Aneurysm of the abdominal aorta.	Crohn's disease

Peripheral artery disease	Premature aging of the skin
Ischaemic heart disease	Loss of smell and taste
Angina pectoris	Decrease in bone density (women)
Haematopoietic cancers	Gangrene
Emphysema	Impotence
Chronic bronchitis	infertility
Pneumonia	

COPD

In clinical practice, COPD is defined by its characteristically low airflow on lung function test. In contrast to *asthma*, this limitation is poorly reversible and usually gets progressively worse over time.

It includes all of the following

1. Chronic bronchitis
2. Emphysema
3. Chronic airway obstruction
4. Chronic non-specific lung disease
5. Non-reversible obstructive airways disease
6. Small airway disease(obliterative bronchiolitis)
7. Cases of chronic asthma with fixed airway obstruction

Risk factors [2]

Exposures

1. Tobacco smoking
2. Biomass solid fuel fires
3. Coal miners and who work with cadmium
4. Outdoor and indoor pollution
5. Childhood infections and maternal smoking—affect lung growth reduce maximally attained lung function in adult life.

6. Recurrent infection – accelerate decline in FEV1. Persistence of adenovirus in lung tissue alter local inflammatory response
7. Low birth weight – reduce maximally attained lung function

Host factors

1. Genetic factor like alpha 1 – antiproteinase deficiency
2. Airway hyper-reactivity

Cigarette smoking :

The major risk factor for mortality from chronic bronchitis and emphysema is cigarette smoking. There are numerous studies which show a significant reduction of FEV_1 (ie) the volume of air exhaled within the first second of the forced expiration in a dose response relationship to the intensity of cigarette smoking which is expressed as pack years. (average number of packs of cigarettes smoked per day multiplied by the total number of years of smoking.) Thus, cigarette smoking is a significant predictor of FEV_1 which is very much declined in COPD.

Pathology : [2]

- Cigarette smoke exposure can affect both large & small air way (< 2mm diameter) & alveoli.
- Changes in large airways cause. Cough & Sputum.
- Changes in small airways & alveoli are responisble for physiological alteration.

- Emphysema & small airway pathology – both are present in most persons with COPD.

Large airway :

Smoking leads to mucous gland enlargement and goblet cell hyperplasia leading to cough and mucus production.

Bronchi can also undergo squamous metaplasia, predisposing to carcinogenesis and disrupting mucociliary clearance.

Neutrophil influx leads to purulent sputum production of upper respiratory tract infections.

Small airways :

Major site of increased resitance in COPD is in airways < 2mm diameter.

Goblet cell metaplasia which replaces the surfactant secreting clara cells and infiltration of mononuclear phagocytes occur.

All these changes lead to luminal narrowing by fibrosis or collapse.

Lung Parenchyma :

- Emphysema is characterized by destruction of gas – exchanging air spaces ie – the respiratory bronchioli alveolar ducts and alveoli.
- The walls of these airways perforate and later obliterate with coalescence into abnormal so much larger air spaces.

- Macrophages accumulate.
- Emphysema is classified into district pathologic types. They are

1. Centriacinar emphysema

- Most frequent type associated with cigarette smoking.
- Characterized by enlarged air spaces in association with respiratory bronchioles.
- Most prominent in the upperlobes and superior segments of lower lobes.
- Often quiet focal.

2. Panacinar Emphysema

- Abnormally large air spaces are evenly distributed in this and across acinar units.
- Seen in patients with $\propto_1$ anti trypsin deficiency.
- It has predilection for the lower lobes.

Pathogenesis :[19]

I.Inhaled noxious particles and gases result in respiratory tract inflammation.

Induce tissue destruction, and impair defense mechanisms

1. Leads to the mucous hyper secretion, increase in goblet cells and inflammatory cell infiltrate --- increased sputum production (leads to chronic bronchitis)
2. narrowing of the airways and leads to fibrosis,
3. Damage to parenchyma of the lungs and
4. Changes in the pulmonary vascular tree and remodelling (leading to impaired cardiac function.)

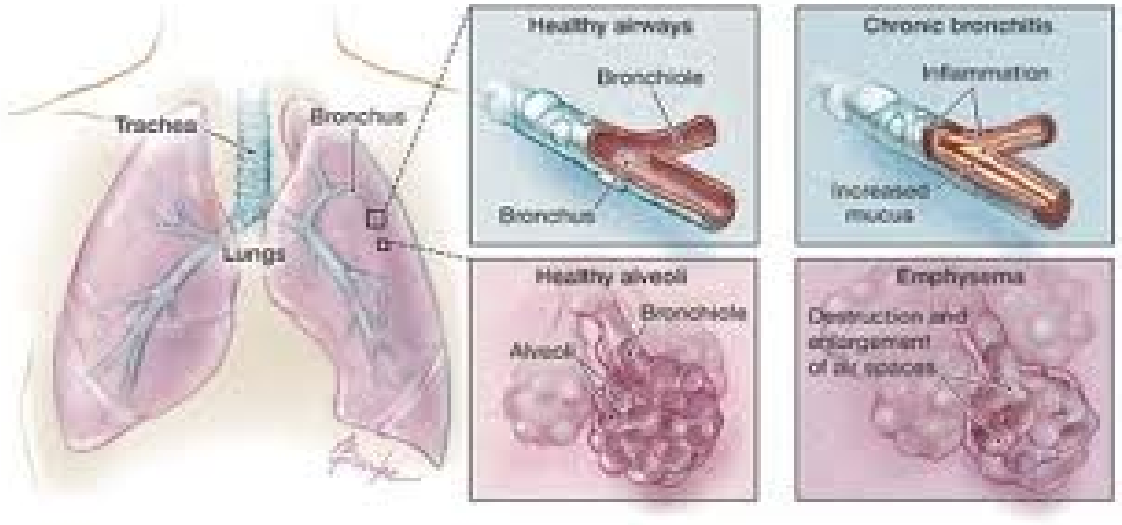
These pathological changes lead to airflow limitation.

Other changes include ----increase in influx of macrophages, neutrophils and T-lymphocytes in most of the parts of the lung .

Release a variety of chemical mediators, many of which (e.g. leukotreine B4, interleukin- 8, and tumour necrosis factor)

Destruction of the lung structures with sustained neutrophilic inflammation.

PICTURE SHOWING THE PATHOLOGY OF COPD :



Another important process involved in the pathogenesis of COPD is an imbalance in the production of elastases and antielastases in the lung, and oxidative stress.

Unopposed action of proteases and oxidants, reactive free radicals

↓

Destruction of alveoli

↓

Appearance of emphysema

Clinical features: [31]

Pulmonary features

- Chronic cough --It is defined as cough for at least 3 months in a year for 2 or more consecutive years which may be intermittent or continuous during the daytime.
- If COPD is associated with cardiac failure,then dyspnoea occur more during nights
- Sputum production –which is of either mucoid or mucopurulent in nature.
- It may be of copious amount in the early morning, especially on getting up from bed.
- Dyspnoea develops during later course of the disease and progresses over time.
- Breathlessness gets worsened on doing exercise and during acute exacerbations of the disease
- Acute exacerbations of COPD
- Repeated episodes of acute bronchitis leads to worsening of symptoms

Most patients would seek medical help only during these episodes of worsening .The systemic features are

1. Muscular weakness due to deconditioning and cellular changes in skeletal muscles
2. Increased circulatory inflammatory markers
3. Impaired sodium and water retention – peripheral oedema
4. Altered lipid metabolism leading to weight loss
5. Increased incidence of osteoporosis

Physical examination :[2]

Physical signs of pulmonary obstruction are rarely present until significant detoriation of lung function has occurred

1. Hyperinflated barrel shaped chest
2. Pursed lips
3. Prolonged expiration
4. Central cyanosis
5. Inward movements of lower ribs and intercostal indrawing during inspiration
6. Palpation --- cardiac apex not palpable
7. Percussion --- loss of cardiac dullness
8. Hyper resonant chest
9. Auscultation --- reduced breath sounds—wheeze
10. Raised JVP , peripheral oedema if cor pulmonale

Severity of COPD GOLD CRITERIA

GOLD Stage	Severity	Symptoms	Spirometry
0	At Risk	Chronic cough, sputum production	Normal
I	Mild	With or without chronic cough or sputum production	FEV_1/FVC <0.7 and FEV_1 80% predicted
II	Moderate	With or without chronic cough or sputum production	FEV_1/FVC <0.7 and 50% FEV_1<80% predicted
III	Severe	With or without chronic cough or sputum production	FEV_1/FVC <0.7 and 30% FEV_1<50% predicted
IV	Very Severe	With or without chronic cough or sputum production	FEV_1/FVC <0.7 and FEV_1<30% predicted or FEV_1<50% predicted with respiratory failure or signs of right heart failure

Abbreviation: GOLD, Global Initiative for Lung Disease.

Source: From RA Pauwels et al.

GOLD Executive Summary – updates

The newly revised document emphasizes the following facts :

1. COPD is characterized by chronic airflow limitation and a range of pathologic changes in the lung.
2. The spirometric classification of COPD includes 4 stages.
    - Stage I – mild
    - Stage II – Moderate
    - Stage III – Severe
    - Stage IV – Very severe
3. It recommends the use of fixed post – bronchodilator FEV, / FVC ratio of < 0.7 to define airflow limitation. But this can not be applied to elderly patients because the normal process of aging affects lung volumes.
4. The Burden of COPD is about 15- 25% in adults of age more than 40 years. They may show airflow limitation of stage I COPD or more than it.
5. Cigarette smoking is a definite risk factor and elimination of this risk factor can prevent and control COPD.

6. COPD should be managed in four ways :[31]
   - Assess and monitor disease.
   - Reduce the risk factors.
   - Manage stable COPD.
   - To manage acute exacerbations.
7. Definition of Acute exacerbation " An event in the natural course of the disease which is characterized by a change in the patient's dyspnoea, cough, and / or sputum which is beyond normal day to day variations, which is acute in onset, and may indicate a change in regular medication in a patient with underlying COPD.

Classical phenotypes

Pink puffers	-	Thin and breathless
		May over-ventilate
		Maintain normal PaCO2 until the late stages
Blue bloater	-	Tolerate / develop hypercapnia earlier
		Abundant purulent sputum
		May develop oedema and secondary polycythemia

Comparison of Two Types of Chronic Obstructive Pulmonary Diseases.

	Predominantly Chronic Bronchitis (Blue – Bloater)	**Predominantly Emphysema (Pink – Puffer)**
Predominant Symptom	Cough	Dyspnoea
Sputum	Copious and purulent	Scant and mucoid
Episodes of bronchial infection	More frequent	Less frequent
episodes of respiratory insufficiency	Frequent	Often terminally
CXR	Increased BV markings at lung bases, large heart	Hyperinflation, bullous changes, small and tubular heart.
Lung compliance	Normal	Increased.
Airway resistance	High	Normal to slight increase.
Arterial blood gases	Abnormality early in course of disease.	Normal until late
Pulmonary Hypertension	Moderate to severe	None to mild
Chronic Cor Pulmonale	Common	Rare
Cardiac Failure	common	Rare

Complications of COPD :[31]

1. Pneumothorax :

The commonest cause for secondary spontaneous pneumothorax is COPD. It may be due to rupture of a bleb or can occur during an acute exacerbation.

It leads to respiratory failure which is life threatening. It should be treated as an emergency, since the condition is curable. Treatment is by doing a Thoracostomy. In patients with advanced COPD, recurrence is common. So they should be treated b;y doing pleurodesis.

Cor Pulmonale:

Chronic alveolar hypoxia leads to the development of Pulmonary hypertension, Right ventricular failure and consequently corpulmonale. Right heart failure signs appear later in the course. After development of cor pulmonale, the median surrival period is diminished. When the pulmonary artery pressure is more than 45mm of Hg, the average survival rate is diminished to less than 2 years.

The condition is treated with continous oxygen therapy for hypoxemia and diuretics for peripheral edema.

Supra ventricular Arrhythmias :

It is common in COPD.

Causes are due to

1. Right atrial enlargement.
2. Increased sympathetic tone.
3. Hypoxemia.
4. Iatrogenic due to drugs like theophyllire and ipratropium.

Hypercapnia :

It is an adaptive response. It is due to decreased work of breathing to reduce the fatiguability of respiratory muscles. It leads to decreased sensation of breathlessness.

The adverse effects are alveolar hypoxia and pulmonary hypertension.

Treatment is to give oxygen in controlled concentrations with venturi masks.

Death in COPD :

Due to

1. Respiratory acidosis and coma
2. Massive collapse of the lungs secondary to pneumothorax
3. 3.Right heart failure.

Investigations :[31]

Two main investigations are X Ray chest and spirometry.

A) X Ray chest :

There are 4 criterias to diagnose emphysema in chest X Ray. They are

a. The retrosternal space should be more than 2.54 cm in lateral radiograph.
b. Hypertranslucency of pulmonary vessels.
c. Low and flat diaphragm below 10^{th} posterior intercostal space.
d. One or more bullae.

Atleast 2 Criteria should be present to diagnose COPD.

B). Spirometric measurement of FEV_1, and FVC is diagnostic of COPD.

It also can assess the severity and progression of the disease.

The hallmark of COPD is airflow obstruction.

Pulmonary function test shows airflow obstruction with a reduction in FEV_1 and FEV_1 / FVC.

With worsening of the disease, lung volumes may increase, resulting in an increase in total lung capacity functional residual capacity and residual volume.

In patients with emphysema, the diffusing capacity will reduce, which reflects the destruction of the lung parenchyma.

The degree of airflow obstruction is an important prognostic factor in COPD.

More recently, a multifactorial index incorporating airflow obstruction, exercise performance dyspnoea and body mass index is a better predictor of mortality rate than spirometry abone.

Arterial Blood gases and oximetry can demonstrate resting or exertional hypoxemia.

The charge in P_H with Pco_2 is 0.08 units / 10 mm Hg acutely and 0.03 units / 10 mm Hg in the chronic state.

Determination of arterial P_H allows the classification of ventilatory failure, defined as $Pco_2 > 45$mm Hg, into acute or chronic states.

Elevated haematocrit indicates the presence of chronic hypoxemia as does the presence of sign of right ventricular hypertrophy.

Computed Tomography is the current definitive test for establishing the presence or absence of emphysema.

Recent guidlines suggest testing for $\propto_1$ Antitrypsin deficiency in all persons with COPD with chronic airflow obstruction.

Differential Diagnosis :

1. Bronchial Asthma –

Usually the patients are young, not a smoker with h/o atopy. Family members can have similar illness – wheezing is the main complaint. Broncho dilators andcortico steroids give excellent relief. Corpulmonale is rare.

2. Congestive Cardiac Failure:

B/L crepitations present on auscultation. Chest X Ray may show cardiomegaly with pulmonary edema. Spirometry shows volume restriction, but there will not be any airflow limitation.

3. Bronchiectasis :

Copious amount of purulent sputum which is most frequently associated with bacterial infections. Coarse leathery crackles will be present on auscultation.

4. Tuberculosis :

Can occur in all ages. CXR will show lung infiltrates. Sputum have to examined for AFB.

5. Obliterative bronchiolitis:

Young age of onset in non smokers. May give h/o rheumatoid arthritis or exposure to fumes. CT may show hypodense areas.

6. Diffue panbronchiolitis :

Most patients are non smokers with h/o chronic sinusitis. CXR and HRCT shows diffuse small centrilobular nodular opacities and hyperinflation.

Difference between Bronchial Asthma and COPD

Bronchial Asthma	COPD
Patient is relatively young.	Middle age or older.
Non smoker	Invariably smokers.
H/o Atopy or family history	Atopy is not essential.
Inbetween symptom free.	Symptoms are invariably persistent and more in winter.
Wheezing is the main symptom	Cough, sputum and dyspnoea are predominant symptom.
Response to bronchodilators and sterodis – excellent.	May not be excellent.
Eosinophilia, sputum eosinophils and positive skin tsts.	Unusual.
Corpulmonale unusual	Very common.
Hypercarbia and Hypoxia – uncommon	Common
Reversibility to bronchodiators is charecteristic.	Some reversibility present, but not more than 20%
Diffusing capacity normal.	Low.
Prognosis – usually good.	Down hill course.

Excluding Alternate Diagnosis:

Tuberculosis should be excluded in all patients with complaints of chronic cough. So sputum should be examined for AFB for a minimum of three times.

Other conditions like fibrocavitatory lesions of TB, bronchiectasis and mass lesions of lungs can be identified by chest X-Ray.

It can also diagnose the other complications like corpulmonale, pneumothorax and bronchopneumonia.

If Bronchial Asthma is suspected, it should be ruled out by doing spirometry. Bronchodilator reversibility test is the best method to exclude bronchial asthma. If spirometry is not available, PEFR with reversibility can be done.

Glucocorticoid reversibility test should be performed in patients with inadequate response to therapy or in patients with suspicion of asthma. Oral prednisolone should be given for 2 weeks and spirometry should be repeated. The criteria for reversibility are

1. Increase in FEV_1 by 200ml.
2. Increase of 15% above baseline.

Confiming the Diagnosis :

The gold standard test for confirmation of COPD is spirometry. It measures FVC, FEV_1 and ration of FEV_1/FVC. The presence of post bronchodilator FEV_1 < 80% of the predited value in combination with FEV_1 / FVC ratio < 70% confirms the presence of air flow limiation which is not fully reversible.

Treatment:[31]

a. A.Non pharmacological method
   - Cessation of smoking
   - Avoiding precipitating factors
   - Pulmonary rehabilitation

b. Pharmacological method
   - Bronchodilators
   - Corticosteroids
   - Theophylline and aminophylline

c. Surgical intervention
   - Bullectomy
   - Lung volume reduction surgery
   - Lung transplantation

d. Others

- Offering vaccination
- Palliative care

Smoking cessation :

It can be attained by

1. Non pharmacological
2. Pharmacological

Non pharmacological

- Educating the patient about the hazards of smoking
- Group counselling
- Hypnosis

Drug therapy:

- Nicotine replacement therapies -- in the form of transdermal patch , lozenges etc.
- Bupropion;
- Nicotine replacement therapyavailable as gum, transdermal patch, inhaler, and nasal spray; and
- Varenicline a nicotinic acid receptor agonist/antagonist

Avoidance of precipitating factors

- Masks can be weared at work place where dust is generated.
- Use of water to suppress dust
- Avoiding open burning of crop residue

Drug treatment :

a. Bronchodilators :[31]

It is central and most important drug used in the symptomatic management of COPD.

Inhaled drugs are usually preferred to oral preparations.

Short acting bronchodilators can be used prophylactically to relieve intermittent or worsening symptoms and when used on regular basis it can prevent or reduce the severity of persistent symptoms.

Regular treatment with short-acting bronchodilators is even though cheaper but it is less convenient for usage than long-acting bronchodilators

The long acting inhaled beta agonist salmeterol can improve the symptoms significantly in doses of 50µg twice daily

In addition if theophylline is given along with bronchodilators or anticholinergics (ipratropium bromide and tiotaropium) it may give an additional improvement.

Step by Step pharmacological therapy :

I. Mild, variable symptoms :

Selective Beta – 2 agonist metered dose inhaler (M D I) aerosol, 1 - 2 puffs every 2 – 6 hours as needed. Maximum dose 8 – 12 puffs over 24 hours.

II. Mild to moderate continuing symptoms :

- Ipratropium MDI aerosol
- 2 to 6 puffs / 6 – 8^{th} hourly.
- Plus Selective Beta 2 agonist
- MDI aerosol 1- 4 puffs / 6^{th} hourly.

III. If no response :

Add sustained release theophylline, 200 – 400mg b.d or 400 – 800mg at bed time for nocturnal broncho spasm And / or Sustained release salbutamol 4 – 8mg bd or bed time only. Mucolytics can be added.

IV. If control of symptoms are not optimal :

- Course of prednisolone 40mg / day for 10 14 days.
- If improvement occurs, taper the drug.
- If no improvement, it can be stopped abruptly and inhaler steroid should be considered.

V. Severe acute exacerbation :

Increase β_2 agonist dose (MDI with spacer 6-8 puffs every ½ - 2 hours or subcutaneous injection of adrenaline or salbutamol or terbutaline in the dose of 0.1 – 0.5 ml. And / or

Increase ipratropium dosage (MDI) 6-8 puffs / 3-4th hourly or inhalation every 4-8 hours and Intravenous theophylline to make serum level of 10-12 mg /ml and Intravenous methyl prednisolone 50-100mg, repeated 6-8 15 hourly should be tapered as early as possible and hydrocortisone should be substituted. Add Antibiotics and mucolytics if indicated.

b. Corticosteroids

Inhaled corticosteroids cannot change the rate of decline in pulmonary function, but can

- increase postbronchodilator FEV_1
- reduce the number of occurrence of acute exacerbations
- slow the rate of decline in morbidity.

It is indicated for

- patients with a documented evidence of spirometric response of reversibility to glucocorticosteroids
- FEV_1<50% of the predicted value.
- Patients with frequent acute severe exacerbations

Their use has been associated with several side effects like

increased rates of oropharyngeal candidiasis and an increased rate of osteoporosis.

Common side effects :

- Loss of bone mineral density,
- weight gain,
- cataracts of the lens,
- glucose intolerance,
- risk of serious and recurrent infection,

If the dose of steroids are reduced and tapered to less thas 10mg/day,then the side effects can be minimized.

c. Oxygen

- Supplemental O_2 is the only pharmacologic therapy demonstrated to unequivocally decrease mortality rates in patients with COPD.
- For patients with resting hypoxemia (resting O_2 saturation 88% or <90% with signs of pulmonary hypertension or right heart failure), the use of O_2 has been demonstrated to have a significant impact on mortality rate
- Patients meeting these criteria should be on continual oxygen supplementation, as the mortality benefit is proportional to the number of hours/day oxygen is used.

Exacerbations of COPD:[31]

- Exacerbations are a prominent and quiet often feature seen frequently in the natural history of COPD.
- Exacerbations are frequent episodes associated with increased breathlessness and cough with change in the amount and character of sputum.
- Sometimes they are associated with fever, sore throat and malaise.
- The frequency of exacerbations increases as airflow obstruction increases;
- Patients with moderate to severe airflow obstruction [GOLD stages III, IV (Table 260-1)] on average have one to three episodes per year.
- However, some individuals with very severe airflow obstruction do not have frequent exacerbations;
- The history of prior exacerbations is a strong predictor of future exacerbations.

Indication for hospitalization of COPD :

Hospitalization :

1. Acute exacerbation with one or more of the following :
    - Inadequate response to outpatient management.
    - Inability to eat or sleep due to dyspnoea.
    - Inability to walk between rooms.

- When patient cannot be managed at home.
- High risk comorbid conditions.
- Altered sensorium.
- Worsening hypoxemia.
- New or worsening hypercarbia.

2. New or worsening of corpulmonale which is unresponsive to outpatient management.
3. During surgery which require sedatives that may worsen the lung function.
4. Comorbid conditions like severe steroid induced myopathy, compression fracture of the vertebra which can worsen the lung functions.

ICU Admission :

1. Severe dyspnoea that do not respond adequately to initial emergency therapy.
2. Confusion, lethargy or respiratory muscle fatigue.
3. Persistent or worsening hypoxemia inspite of O_2 therapy.
4. When mechanical ventilation is required.
5. General approach to management of COPD :
6. Establish the diagnosis and assessment of symptoms.
7. Cessation of smoking should be achieved completely.

8. Encourage healthy life style pattern, exercise and immunization.
9. Treatment of obstruction by drugs.
10. Assessment of hypoxemia, O_2 therapy given if needed.
11. Assess the response to therapy.
12. Referral to higher centre if necessary.
13. Patient education.

Management of acute exacerbation:[31]

Bronchodilators :

Patients are treated with an inhaled agonist, often with the addition of an anticholinergic agent.

The frequency of administration depends on the severity of the exacerbation.

Patients are treated initially with nebulized therapy, as such treatment is often easier to administer in older patients or to those in respiratory distress.

The addition of methylxanthines (such as theophylline) to this regimen can be considered.

Antibiotics :

Patients with COPD are frequently colonized with potential respiratory pathogens.

Bacteria frequently implicated in COPD exacerbations include

- *Streptococcus pneumonia*
- *Haemophilus influenza*
- *Moraxella catarrhalis*.

In addition, *Mycoplasma pneumoniae* or *Chlamydia pneumoniae* are found in 5-10% of exacerbations.

The choice of antibiotic should be based on local patterns of antibiotic susceptibility of the above pathogens as well as the patient's clinical condition.

Glucocorticords

The use of glucocorticoids has been demonstrated to reduce the length of stay, hasten recovery, and reduce the chance of subsequent exacerbation or relapse for a period of up to 6 months.

A study demonstrated that 2 weeks of glucocorticoid therapy produced benefit indistinguishable from 8 weeks of therapy.

The GOLD guidelines recommend 30–40 mg of oral prednisolone or its equivalent for a period of 10–14 days.

Hyperglycemia, particularly in patients with preexisting diagnosis of diabetes, is the most frequently reported acute complication of glucocorticoid treatment.

Oxygen

Supplemental O_2 should be supplied to keep arterial saturations 90%

Hypoxemic respiratory drive plays a small role in patients with COPD.

Studies have demonstrated that in patients with both acute and chronic hypercarbia, the administration of supplemental O_2 does not reduce minute ventilation.

It does, in some patients, result in modest increases in arterial P_{CO2}, chiefly by altering ventilation-perfusion relationships within the lung.

This should not deter practitioners from providing the oxygen needed to correct hypoxemia.

Indications for long term oxygen therapy :

I. Continous Oxygen :

Resting $P_a O_2$ of 55mm of Hg or less or oxygen saturation of 88% or less.

Resting $P_a O_2$ of 56-59mm Hg or oxygen saturation of 89% in the presence of any of the following :

- Dependent edema suggesting cardiac failure.
- ECG showing P pulmonale.
- Polycythemia.

II. Non continous oxygen :

During exercise – Resting $P_a O_2$ of 55mm Hg or less or oxygen. Saturation of 88% or less with a low level of exertion.

During Sleep :

Resting $P_a O_2$ < 88% with associated complications like pulmonary hypertension, day time somnolence and cardiac arrythmias.

Mechanical ventilatory support

When Pa_{CO2}>45 mmHg, patient is said to be in respiratory failure and immediate initiation of non invasive positive pressure ventilation is recommended. It significantly leads to a reduction in mortality rate.

Indications for NIPPV :

Selection Criteria :

- Moderate to severe dyspnoea with use of acessory muscles and paradoxical abdominal movements.
- Moderate to severe acidosis of P_H < 7.35 and / or hypercapnia of Pa Co_2 > 6 K pa, 45mm of Hg.
- Respiratory rate > 25 / minute.

Indications for invasive mechanical ventilation :

- Unable to tolerate NIPPV or NIPPV failure.
- Severe dyspnoea with use of accessory muscles and paradoxical abdominal motion.

- Respiratory Rate > 35 / minute.
- Life threateing hypoxaemia.
- Severe Acidosis P_H < 7.25 and / or hypercapnia Pa Co_2 > 8 k Pa,60mm Hg.
- Respiratory arrest.
- Worsening in mental status despite optimal therapy.
- Complications like shock, hypotension.
- Other compliations like electrolyte, imbalance, sepsis, pneumonia, pulmonary embolism, barotrauma, massive pleural effusion.

Discharge criteria for patients with COPD exacerbations :

Inhaled β2 agonist therapy is required no more frequently than every 4 hourly.

Patient is able to walk across the room.

Patient is able to eat and sleep without frequent awakening by breathlessness.

Patient has been clinically stable for 12 – 24 hours.

Patient and attenders understand the correct use of medicatins and they are confident enough to manage successfully at home.

Follow – up visits :

- Done 4-6 weeks after discharge.
- Measurement of FEV, done.
- Reassessment of inhaler technique.
- Understanding of treatment regimen.
- Need for longterm O_2 therapy and / or home nebulizer espescially for stage IV Patients.

Prognosis of COPD :

If COPD is clinically evident, then the median survival period is only about 10 years.

The factors which are associated with poor prognosis are

1. Low FEV_1
2. Presence of Cor Pulmonale
3. Severe Breathlessness
4. Resting Tachycardia.
5. Hypoxemia
6. Continuos active smoking
7. Poor nutrition
8. Anaemia
9. Poor quality of life
10. Reduced exercise tolerance

11. Frequent Acute Severe exacerbations
12. Co-morbid illness
13. Low DLCO

There is also a multidimensional prognostic index – BODE Index which includes:

1. Body mass index
2. Obstructive ventilatory defect severity
3. Dyspnoea severity
4. Exercise capacity

Score :

1. 7 – 2 year mortality is 30%
2. 5 – 6 – 2 year mortality is 15%
3. < 5 – 2 year mortality i less than 10%

No drug treatment has shown to alter the natural history of COPD. Smoking cessation is the only intervention that can arrest the rapid decline in lung function. Home Oxygen therapy can also improve the prognosis in patients suffering from hypoxemia.

The best guide to the progression of COPD is the change in FEV_1, over time. FEV_1, declines with normal aging at about 30ml / year, but this decline is increased to 45ml/year in smokers. Cessation of smoking will produce only a small improvement in FEV_1, but the subsequent fall

in FEV_1, progressively slows to the rate of 30ml/year, as that of in non smokers.

Depending upon the diease severity, the 5 year mortality rate of patients with COPD is 40-70%. The three major causes of death are COPD itself, Bronchogenic Carcinoma and Corpulmonale. The age and reduction in FEV_1, are the most ommon prognostic factors.

MATERIALS AND METHODS

PLACE OF STUDY

This study was conducted on 50 patients in the general medical ward.

PERIOD OF STUDY

From NOV 2011 to NOV2012.

DESIGN

Observational prospective cohort study of patients who are smokers admitted in the hospital for complaints other than respiratory symptoms. A total of 50 patients were included in the study.

METHODOLOGY

A. Subject selection

1. Inclusion criteria

a. Subjects randomly selected from medical ward in age group 20 -60 years.
b. with h/o smoking of atleast on ten pack years(ie.. > 10 cigarettes or 20 beedies per day for atleast ten years).

2. Exclusion criteria

a. Patients who had symptoms as per American
b. Thoracic Society scale for dyspnoea
c. Known case of cardiac disease.
d. Known diabetic patients.

e. Patients with spinal deformity.

f. Age > 60 years

g. Women

All patients included in the study were subject to thorough clinical examination. All were subject to laboratory investigation as per the profoma.

UDWADIA'S FORMULA

VARIABLES	**MALE**	**FEMALE**
FEV 1(L)	Adults <30years	Adults <30years
	0.039×H-0.010 × A-3.266	0.025× H-0.011 × A-1.424
	Adults >30years	Adults >30years
	0.037×H-0.022× A-2.650	0.032×H-0.012×A-2.580
FVC(L)	Adults <30years	Adults <30years
	0.055×H+0.019×A-6.058	0.030×H+0.006×A-2.284
	Adults >30years	Adults >30years
	0.054×H-0.018×A-4.832	0.043×H-0.010×A-3.755
PEF	0.085×H-0.0187×A-6.2083	0.0497×H-0.0336×A-0.1399
FEF25-75%	0.0173×H-0.0407×A+1.6108	0.0245×H-0.0336×A0.1399
FEF50	0.0195×H-0.0365× A+1.7383	0.0272×H-0.279×A-0.2704
FEF75	0.0088×H-0.0301×A+1.0402	0.0113×H-0.0288×A+0.5012

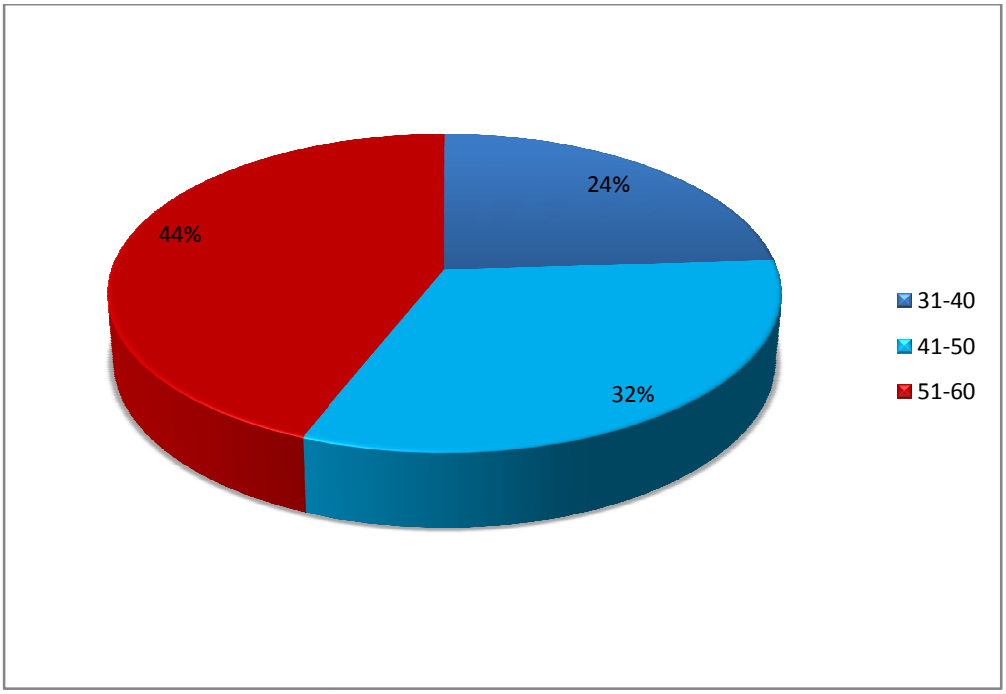
A - Age in years, H - Height in cms

OBSERVATIONS AND RESULTS

Table - 1

Agewise distribution of smokers

Age in years	Frequency	Percent
31-40	12	24.0
41-50	16	32.0
51-60	22	44.0
Total	50	100.0

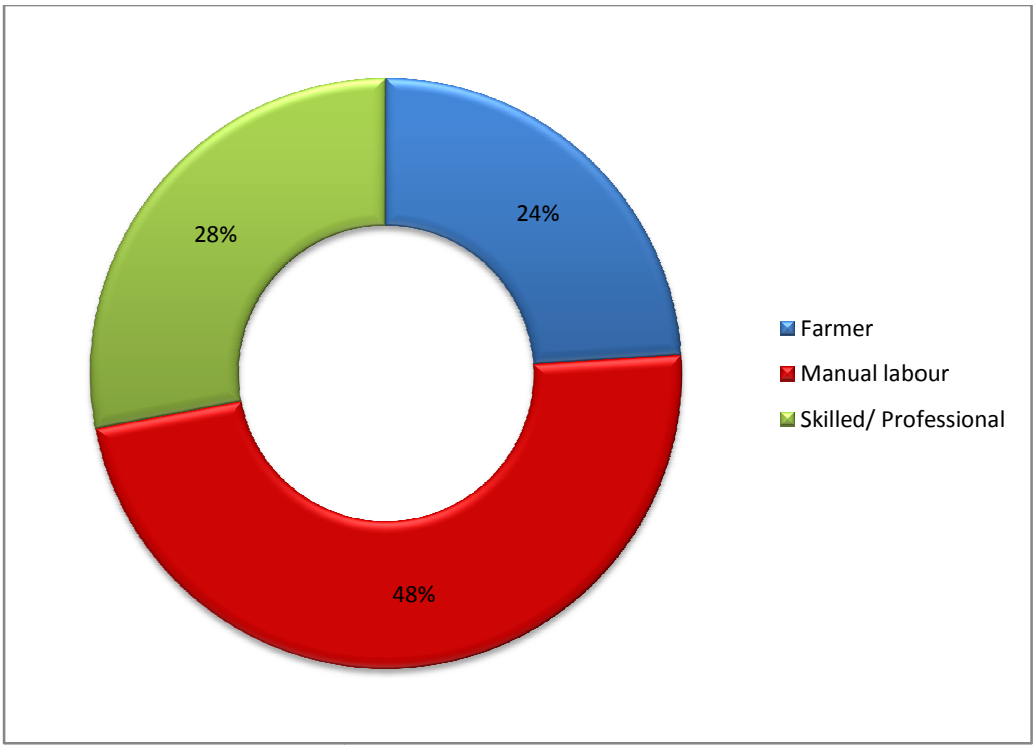


Maximum percentage of smokers in the study belongs to the age group of 51 to 60 years and minimal percentage belongs to 31 to 40 years of age.

Table-2

Smoking and occupation

Occupation	Frequency	Percent
Farmer	12	24.0
Manual labour	24	48.0
Skilled/ Professional	14	28.0
Total	50	100.0

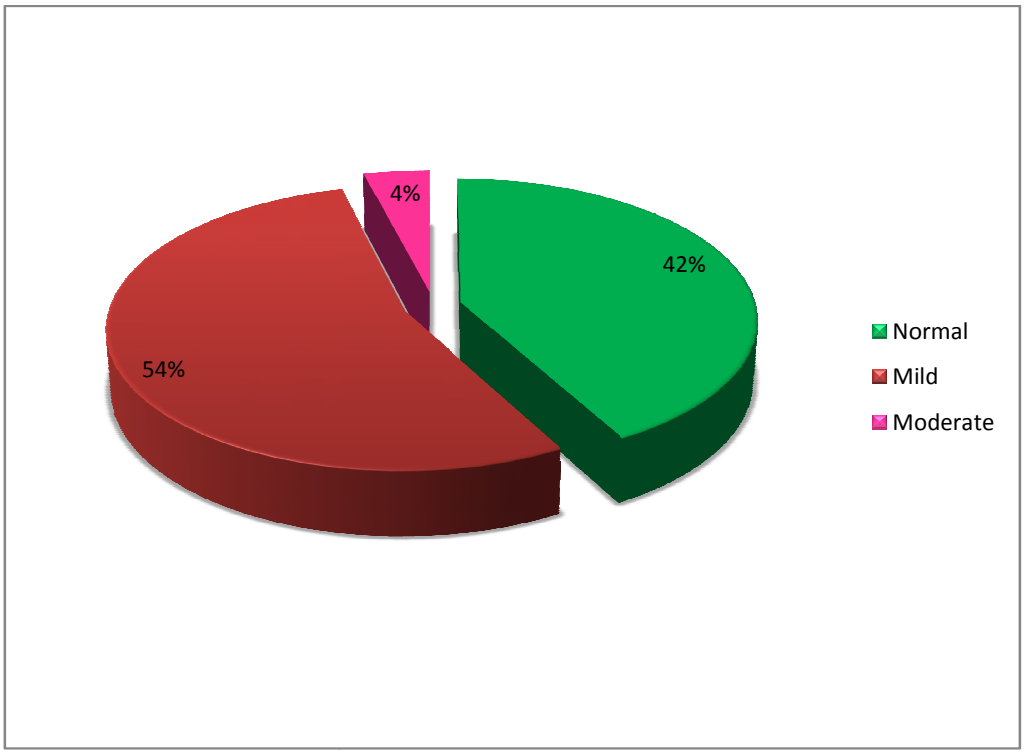


Manual labourers are the group who are associated with increased percentage of smoking in this study.

Table - 3

Degrees of FEV1

FEV1 stages	Frequency	Percent
Normal	21	42.0
Mild	27	54.0
Moderate	2	4.0
Total	50	100.0

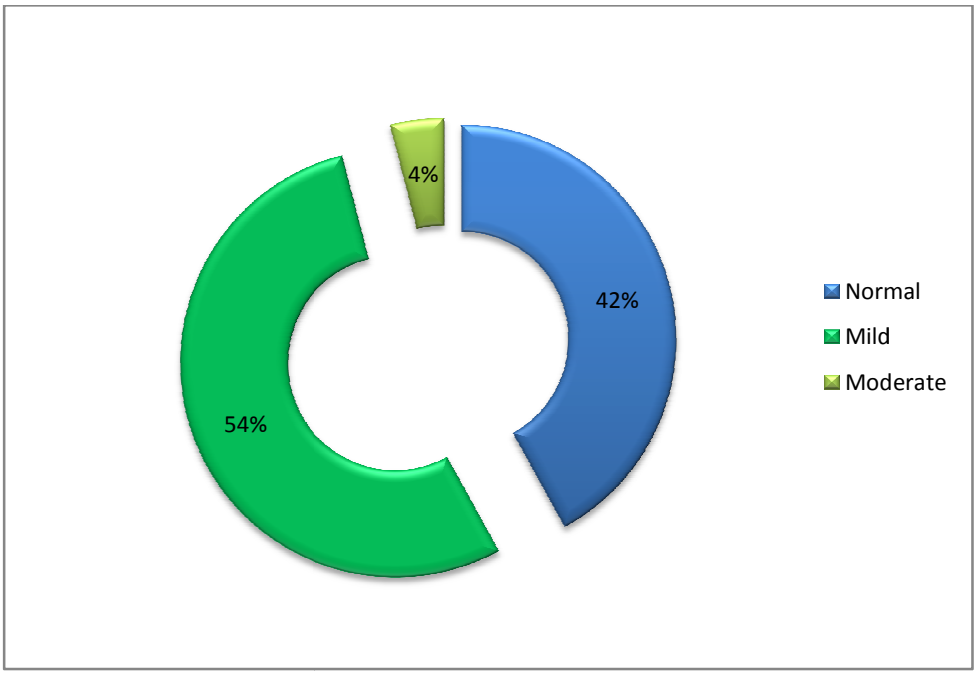


FEV1 is normal in 42%,mildly decreased in 54% and moderately decreased in 4% of the population under study. no one had severe obstruction.

Table - 4

FEV1/FEC ratio in various individuals

FEV1/FVC	Frequency	Percent
Normal	21	42.0
Mild	27	54.0
Moderate	2	4.0
Total	50	100.0

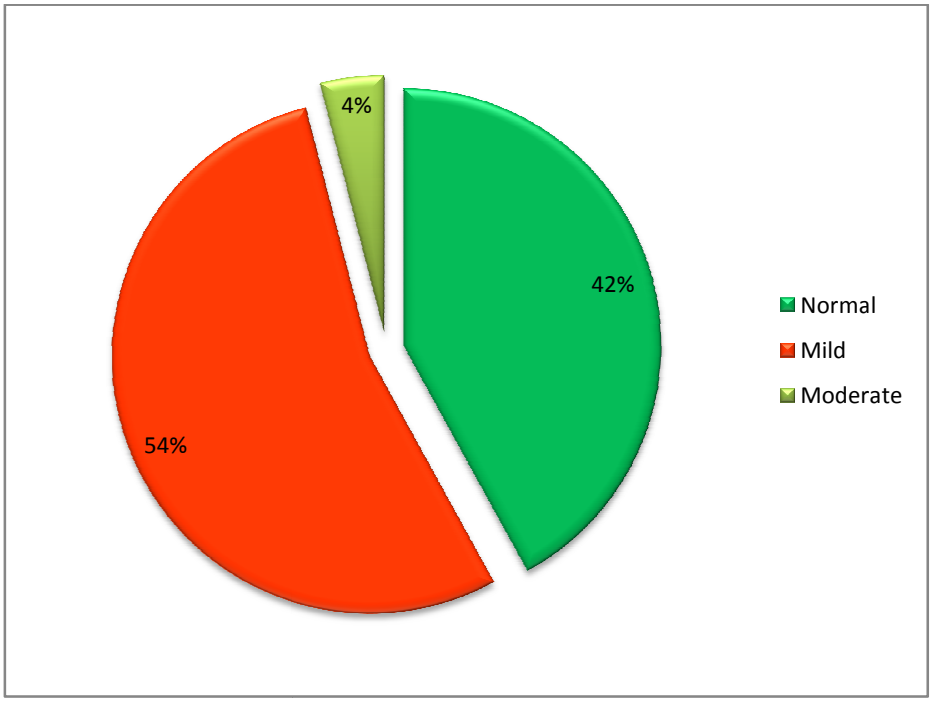


FEV1/FVC ratio is mildly decreased in 54%,moderately decreased in 4% of the population in this study.It is normal in the remaining persons and none showed severe obstruction.

Table - 5

FEF values

FEF	Frequency	Percent
Normal	21	42.0
Mild	27	54.0
Moderate	2	4.0
Total	50	100.0

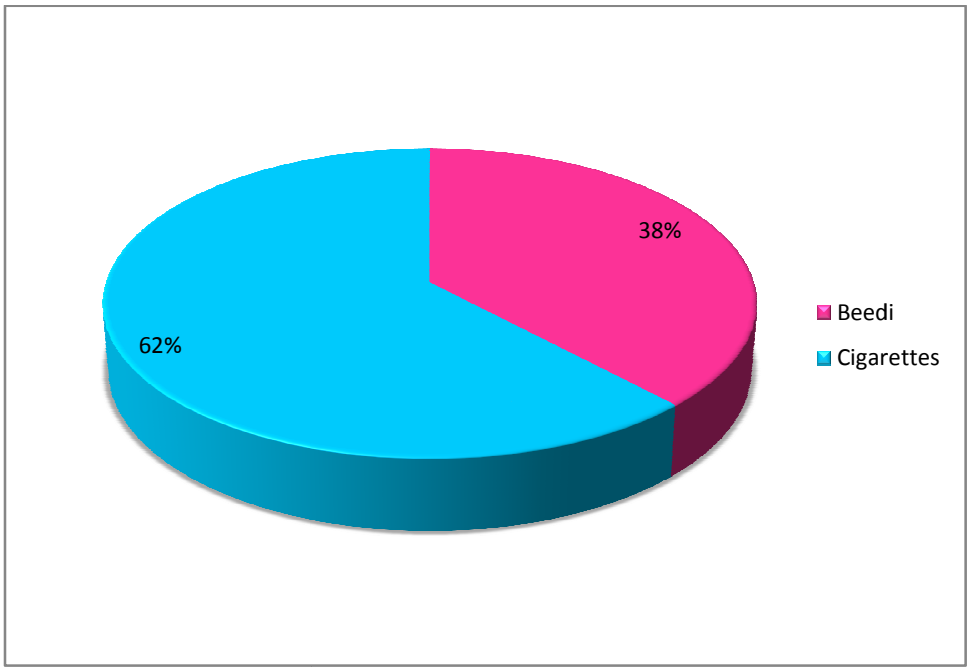


FEF25-75% also showed similar results as that of FEV1 and FEV1/FVC ratio.

Table -6

Types of smoking

Type	Frequency	Percent
Beedi	19	38.0
Cigarettes	31	62.0
Total	50	100.0

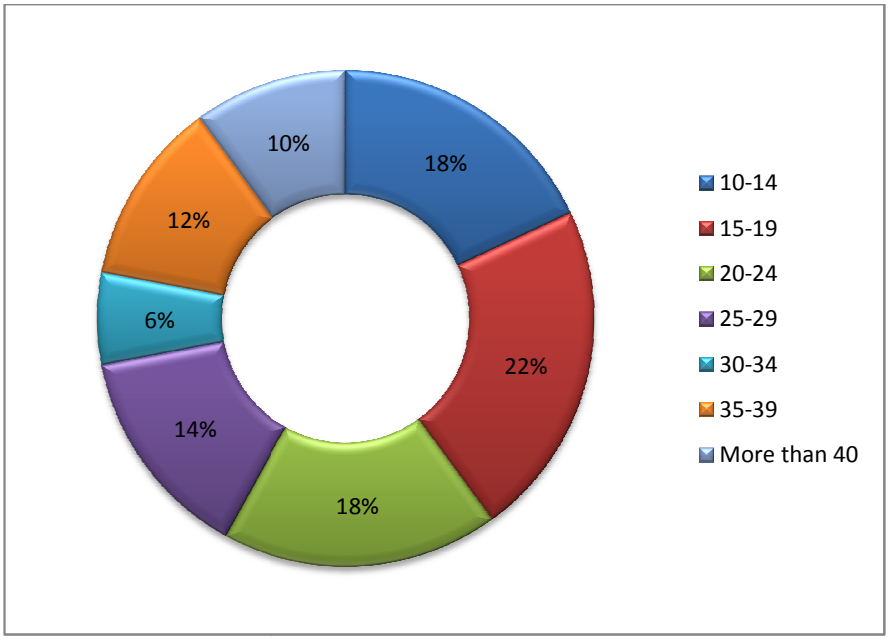


In the study group, about 62% smoked cigarettes and 38% smoked bidis.

Table - 7

showing the percentage of smokers with duration of smoking in years

Duration of smoking in years	Frequency	Percent
10-14	9	18.0
15-19	11	22.0
20-24	9	18.0
25-29	7	14.0
30-34	3	6.0
35-39	6	12.0
>40	5	10.0
Total	50	100.0

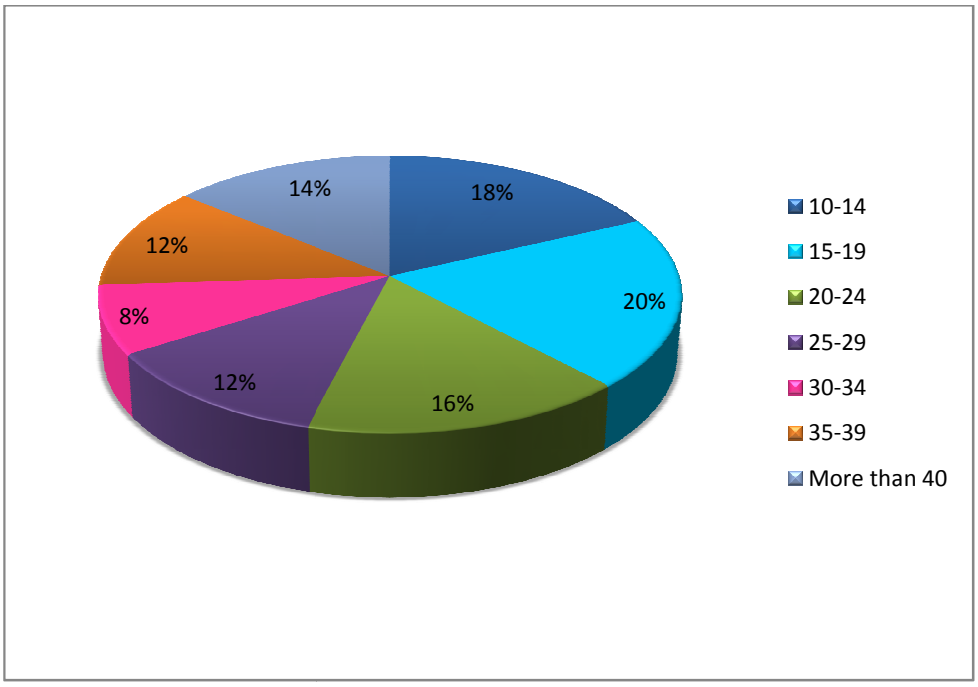


In this study,maximum number of smokers, (i.e) 22% smoked for 15-19 years and only 3% of smokers smoked for a long duration of 30-34 years.

Table – 8

Comparison of percentage of smokers with pack years

Pack years	Frequency	Percent
10-14	9	18.0
15-19	10	20.0
20-24	8	16.0
25-29	6	12.0
30-34	4	8.0
35-39	6	12.0
>40	7	14.0
Total	50	100.0



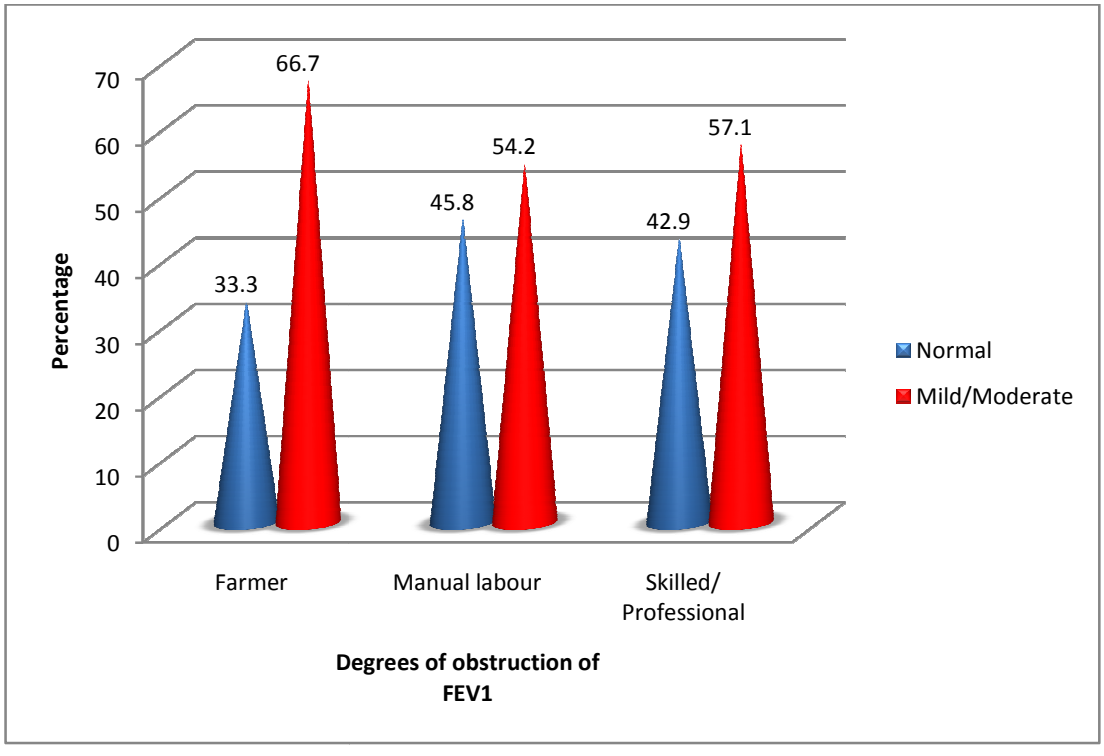
20% of smokers had 15-19 pack years of smoking and 8% had 30-34 pack years.

Table – 9

Comparison of FEV1 reduction in relation to occupation

Occupation	FEV1				Total	
	Normal		Mild/ Moderate			
	N	%	N	%	N	%
Farmer	4	33.3	8	66.7	12	100
Manual labour	11	45.8	13	54.2	24	100
Skilled/ Professional	6	42.9	8	57.1	14	100
Total	21	42	29	58	50	100

χ^2 =0.519 df=2 p=0.771

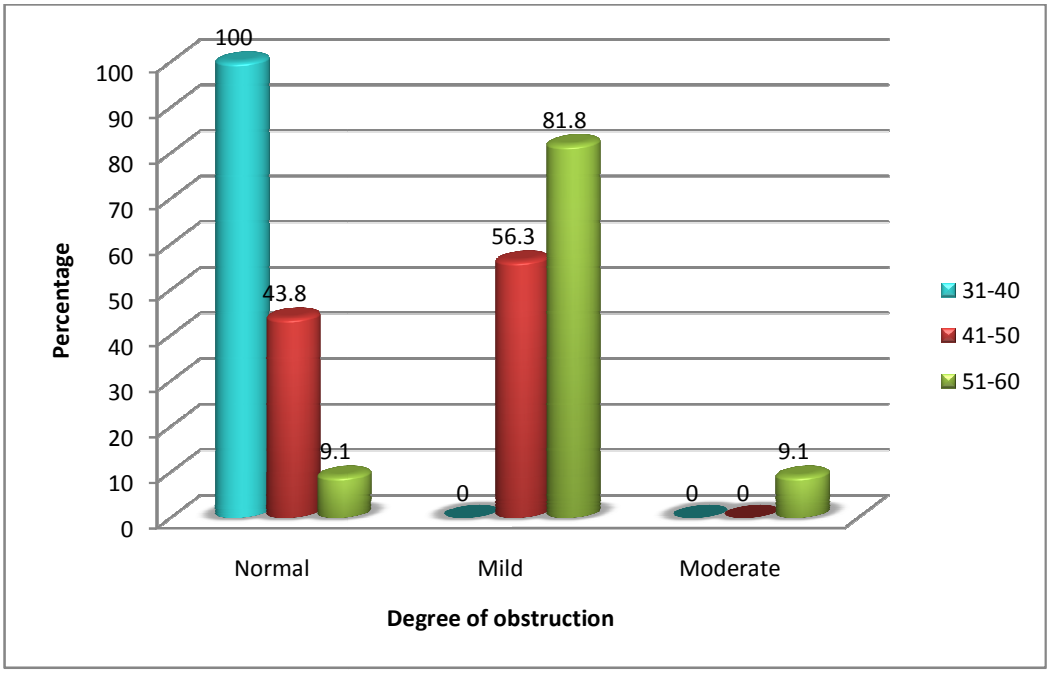


There is no significant association between occupation and reduction in FEV1.

Table - 10

Comparison of FEV1 reduction in relation to age

Age	FEV1						Total	
	Normal		Mild		Moderate			
	N	%	N	%	N	%	N	%
31-40	12	100.0	0	.0	0	.0	12	100.0
41-50	7	43.8	9	56.3	0	.0	16	100.0
51-60	2	9.1	18	81.8	2	9.1	22	100.0
Total	21	42.0	27	54.0	2	4.0	50	100.0



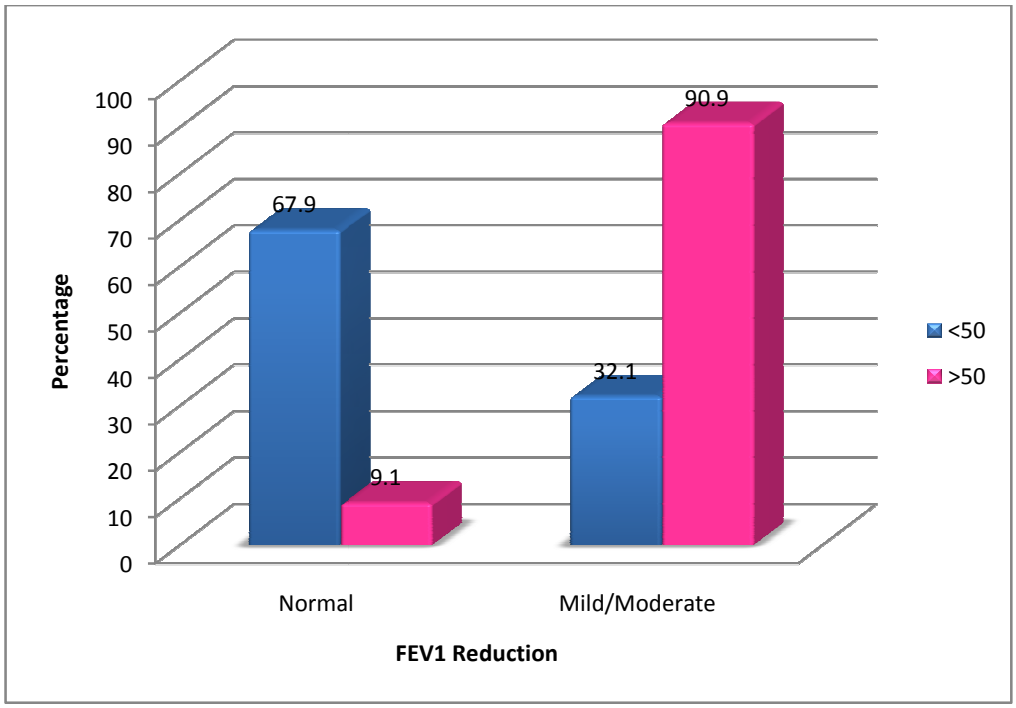
There is a significant association between age and reduction in FEV1.Smokers of the age group 51-60 years had marked limitation in air flow rates.

Table - 11

Comparison of mild and moderate reduction of FEV1 in mild and moderate reduction of FEV1 in relation to age.

Age	FEV1				Total	
	Normal		Mild/ Moderate			
	N	%	N	%	N	%
<50	19	67.9	9	32.1	28	100
>50	2	9.1	20	90.9	22	100
Total	21	42	29	58	50	100

χ^2 =17.466 df=1 p<0.001

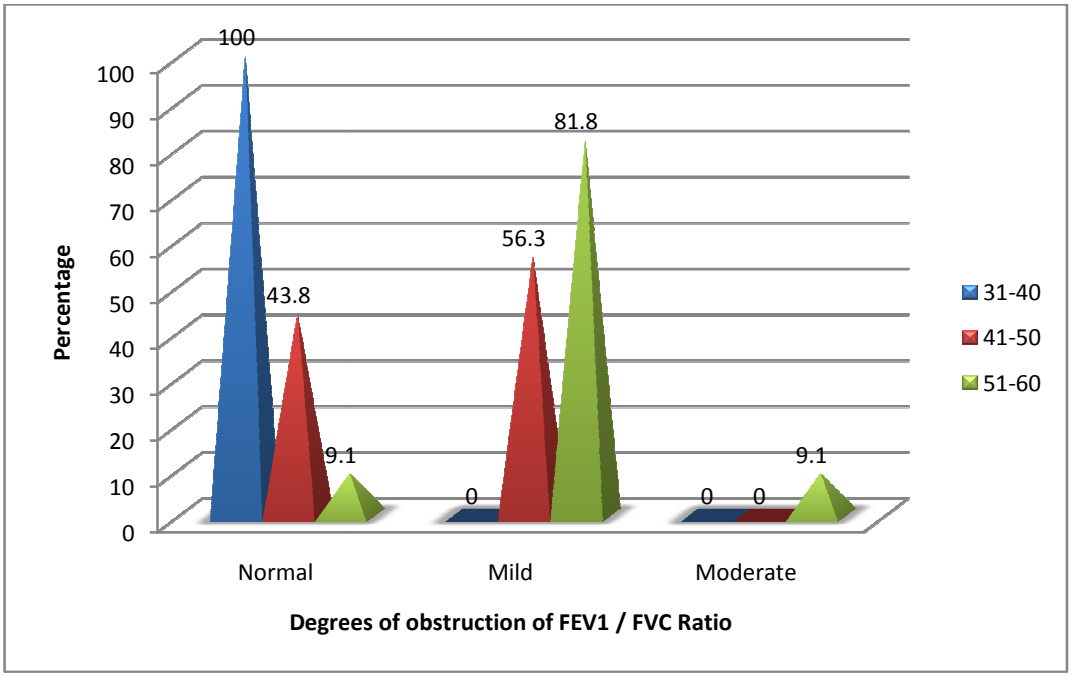


Smokers of more than 50 years of age had significant reduction in FEV1 when compared to smokers of less than 50 years of age. This is very significant.

Table - 12

Comparison of FEV1/FVC ratio in relation to age

Age	FEV1/FVC						Total	
	Normal		Mild		Moderate			
	N	%	N	%	N	%	N	%
31-40	12	100.0	0	.0	0	.0	12	100.0
41-50	7	43.8	9	56.3	0	.0	16	100.0
51-60	2	9.1	18	81.8	2	9.1	22	100.0
Total	21	42.0	27	54.0	2	4.0	50	100.0

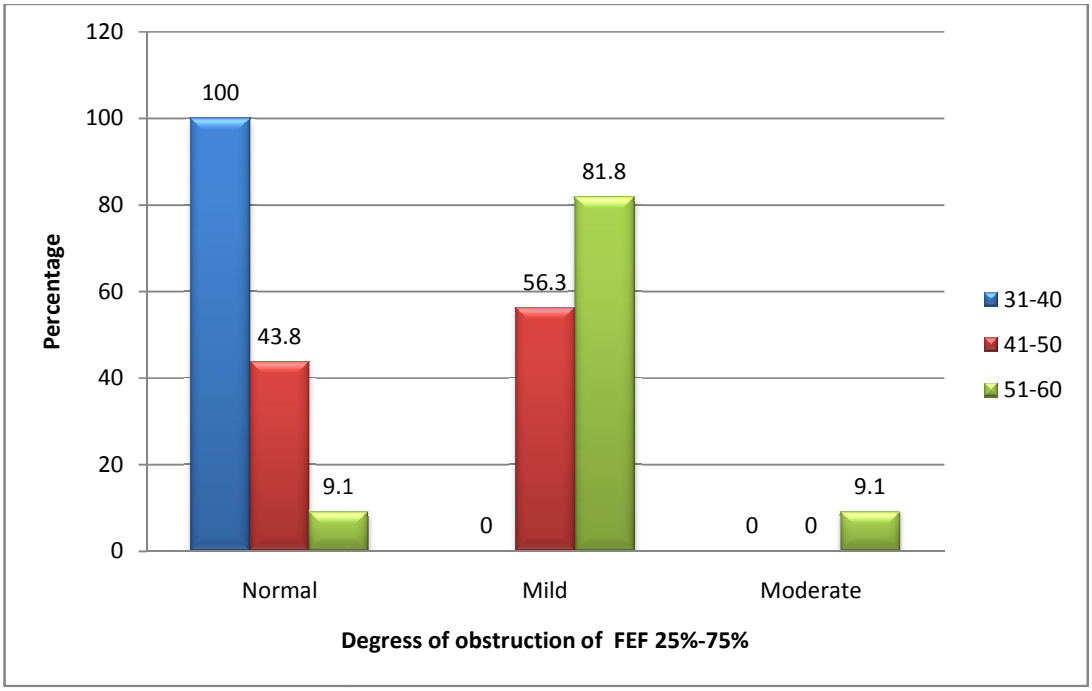


FEV1/FVC ratio is very much declined to mild and moderate degrees in 51-60 years age of smokers.It is declined to a lesser extent in the age group of 41-50 years.

Table - 13

Comparison of reduction of FEF 25%-75% in relation to age

Age	FEF 25-75%						Total	
	Normal		Mild		Moderate			
	N	%	N	%	N	%	N	%
31-40	12	100.0	0	.0	0	.0	12	100.0
41-50	7	43.8	9	56.3	0	.0	16	100.0
51-60	2	9.1	18	81.8	2	9.1	22	100.0
Total	21	42.0	27	54.0	2	4.0	50	100.0



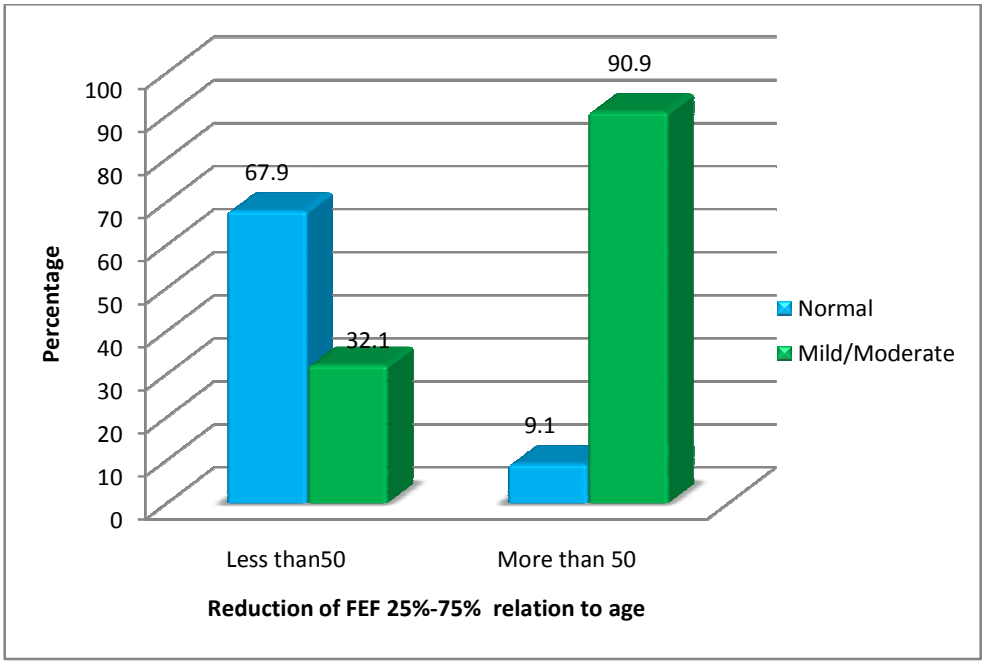
FEF25-75% also shows similar distribution as that of other parameters with the age group under study.

Table - 15

Comparison of mild and moderate reduction of FEF 25%-75% in relation to age

Age	FEF25-75%				Total	
	Normal		Mild/ Moderate			
	N	%	N	%	N	%
<50	19	67.9	9	32.1	28	100
>50	2	9.1	20	90.9	22	100
Total	21	42	29	58	50	100

χ^2 =17.466 df=1 p<0.001



FEF25-75% shows a significant reduction in age more than 50 years when compared with age less than 50 years.

Table – 16

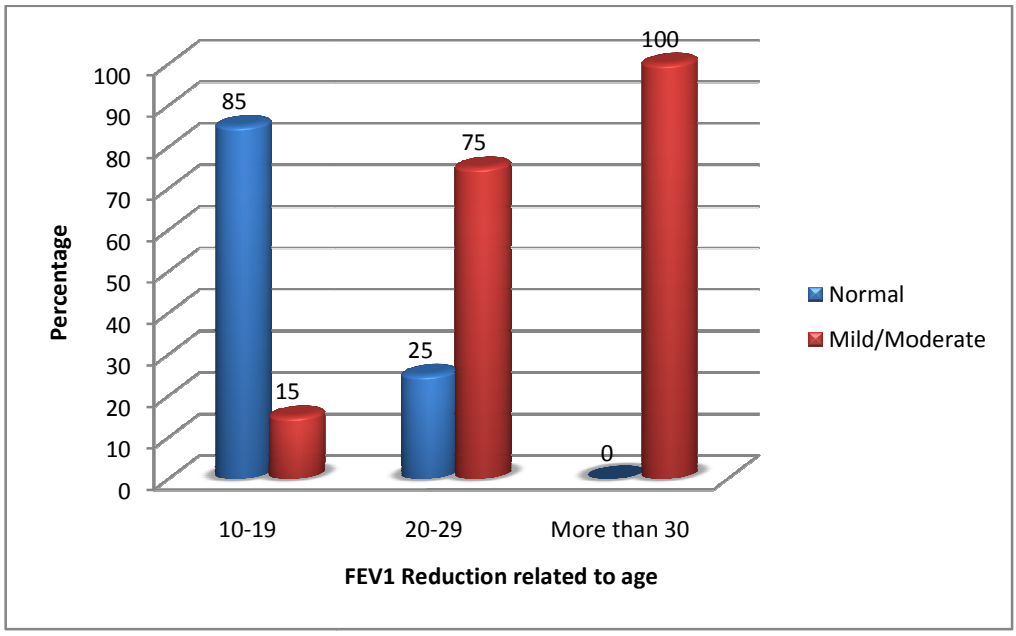
Comparison of FEV1 reduction in relation to duration of smoking in years

Duration of smoking in years	FEV1				Total	
	Normal		Mild/ Moderate			
	N	%	N	%	N	%
10-19	17	85.0	3	15.0	20	100.0
20-29	4	25.0	12	75.0	16	100.0
>30	0	.0	14	100.0	14	100.0
Total	21	42.0	29	58.0	50	100.0

χ^2 =27.217 df=2 p<0.001

FEV1 shows a significant reduction with prolonged duration of smoking. More than 30 years of smoking in this study showed significant airfiow Limitation when compared with 10-19 years of smoking duration.

Comparison of FEV1 reduction in relation to duration of smoking in years



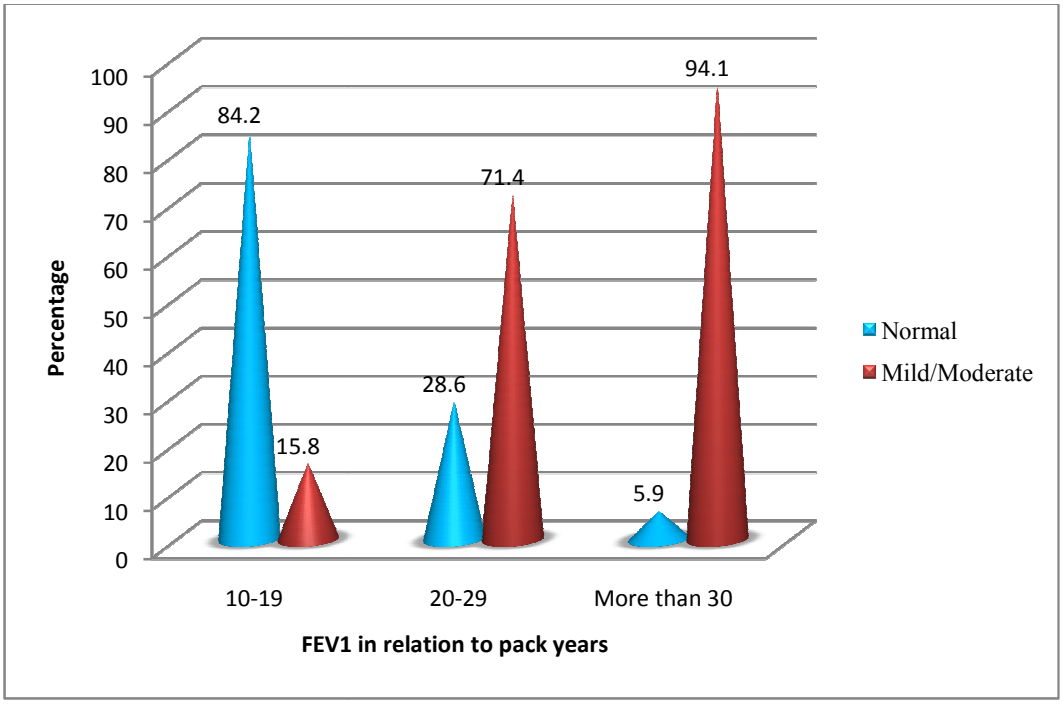
FEV1 shows a significant reduction with prolonged duration of smoking. More than 30 years of smoking in this study showed significant airfiow Limitation when compared with 10-19 years of smoking duration.

Table -17

Comparison of FEV1 reduction in relation to packyears

Pack years	FEV1				Total	
	Normal		Mild/ Moderate			
	N	%	N	%	N	%
10-19	16	84.2	3	15.8	19	100
20-29	4	28.6	10	71.4	14	100
>30	1	5.9	16	94.1	17	100
Total	21	42	29	58	50	100

χ^2 =24.037 df=2 p<0.001



FEV1 reduction is significantly reduced in smokers > 30 pack years When compared to smokers< 30 pack years.

DISCUSSION

In this study, a major group about 54% showed mild obstructive pattern, a minor group of about 4% showed moderate obstructive pattern and the remaining 42% had normal lung function. The multicentre Lung Health study conducted a similar randomized clinical trial by doing spirometry in current smokers, but without any symtoms 25% of the population under study had a moderate airflow obstruction and 5% had severe airflow limitation which was comparable to this study[44].

In this study, there is a significant airflow limitation in relation to the age distribution and pack years of smoking.

In a similar study done by Dan Stanescu et al, the middle aged smokers are at less risk of functional detoriation, but smokers above the age of 50 years were at high risk of development of COPD which was comparable to this study. A similar study was done in Canada which also showed the prevalence of airway obstruction of about 4.6% in the age group of 55 – 64 years, 5% in 65 – 74 years and 6.8% in the age group of >75 years which was comparable to this study.

In this study, the mean FEV1, levels were 2.6 ± 0.6 in smokers with smoking history less than 12 median pack years and 1.8 ± 0.6 in smokers with history of smoking more than 12 pack years. The rate of

decline of FEV1, is very significant with increase in the number of pack years.

Young et al, done a similar study which showed a significant reduction of FEV1, and he used the measure of airflow limitation to assess the risk of development of not only COPD, but also the risk of incidence of bronchogenic carcinoma, coronary arterial heart disease and cerebro vascular accident.

Fletcher CM et al also conducted a similar study in early stages of development of COPD, which showed that FEV1, falls gradually over few years in smokers which was comparable to this study.[45]

In this study, there is a decline of FEF 25-75% in the smokers, which was more in the smokers of longer pack years duration than in smokers of less pack years duration which is very significant. FEF 25-75% is the major predictor of small airway obstruction and therefore the incidence of COPD.

A similar study was done by Pauwells RA et al, which showed significant reduction in FEF 25-75% in smokers which was comparable to this study.[46]

In this study, the FEV, FVC ratio shows mild GOLD airflow limitation in 54% of the smokers, moderate limitation in 4% of smokers which is very significant.

A similar study was done by Polatl Mehmet et al, where there was significant disease in FEV, FEV/FVC ratio and FEF 25-75%, FEF 75% which was comparable to this study[48]

In this study, there is significance with the type of smoking and occupation. The cigarettes were significantly associated with Sedentary occupation where as Beedis were associated with heavy manual workers.

Both Beedis and Cigarette are associated with the development of COPD. Filter tipped cigarettes are less deleterious when compared to Beedis.

Thus spirometry is a very useful screening method to detect the airflow limitation very earlier in the course of incidence of COPD, if it is done during earlier age and pack years of smoking. Identification of even mild degree of air flow limitation is very significant, so that smoking cessation with reduce the burden of the chronic obstructive pulmonary disease to the society.

CONCLUSION

In this study, 42% of smoker, had normal lung function, 58% showed obstructive pattern in their lung functions.

A significant number of patients had small airway disease as evidenced by decreased FEF 25-75%.

A decline in FEV, FEV/FVC ratio were observed in 58% of asymptomatic smokers, who may develop obstructive lung disease in the near future.

Increasing age, increased quantum of smoking both in duration and number which is expressed in pack years are significant confounding factors in the development of airflow limitation.

A definitive conclusion cannot be arrived with the association of smoking and occupation, since only a small sample size was studied.

Routine lung function tests by doing spirometry in smokers without symptoms is an important screening test, so that the incidence of COPD can be detected very earlier in the course of the disease.

Thus spiromety is essential to detect the tip of iceberg of COPD.

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PROFORMA

Case No.

Name : Age : Sex :

IP No. : DOA : DOD:

Address : Occupation :

FINAL DIAGNOSIS:

H/o Hypertension

H/o diabetes mellitus

Past History

IHD ☐ TB ☐ RHD ☐ BA ☐

Personal History

Tobacco use: Smoking Beedi/ Cigarette/ pipe / cigar

Quantity

Period Other forms

Alcohol

GENERAL EXAMINATION

Height Weight BMI

PR : RHYTHM : CHARACTER :

Temperature: BP:

Respiratory rate: Type:

Pallor: Cyanosis:

Lymph node: Clubbing:

JVP:

RESPIRATORY SYSTEM

UPPER RESP.TRACT: NOSE

PNS

THROAT

INSPECTION

Shape of chest: B/L Symmetrical / Not

Trachea position: R / Central / L

Any spinal deformity: Scoliosis / kyphosis / Normal

Any chest deformity: Hollowing / Bulging / Normal

Movement of chest: Equal / Not

Any dilated veins/pulsations: Present / Not

Any scar / sinuses : Present / Not

Use of accessory muscles: Yes / No

Intercostal retraction: Yes / No

Apical impulse: Visualized / Not

PALPATION

Tracheal position: R / Central / L

Movement of chest: Equal / Not

Apical impulse: Normal / Not

MEASUREMENTS

AP Diameter:

Transverse diameter:

Chest circumference:

Expansion:

Palpable rales/ ronchi: Yes / No

Site

Percussion

Anteriorly

Kronig isthmus

Infraclavicular

Mammary

Liver dullness 5th ICS Yes / No

Traube's area Tympanic / Not

Tidal percussion:

Posteriorly

Suprascapular:

Infrascapular:

Interscapular:

Axillary:

Infra axillary:

AUSCULTATION

Breath sounds - normal / diminished

Type - Vesicular / Bronchovesicular / Bronchial

Added sounds – rales / rhonchi

Site :

VR Equal / Not

CVS

ABDOMEN

CNS

Investigations :

Blood Sugar : URINE :

Serum Creatinine :

CXR PA :

ECG :

Echo :

Spirometry :

Others

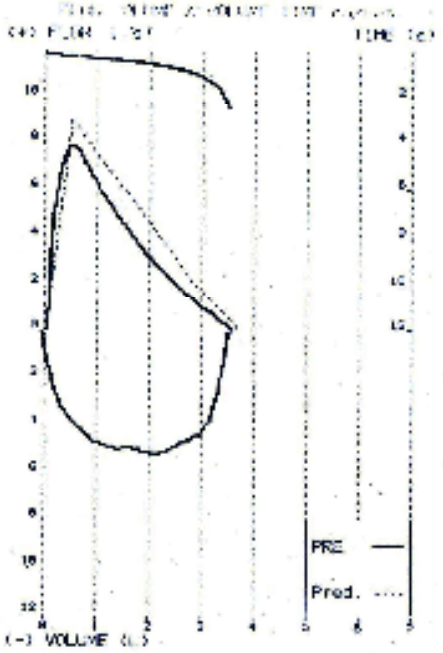
Bronchoscopy

HRCT CHEST

S.NO	NAME	AGE	SEX	OCCUPATION	DURATION OF SMOKING	FEV1	FEV1/FVC	FEF 25%-75%	PACK YEARS	CIGARETTES/BIDIS
1	PONNUSAMY	40	M	FARMER	20 YRS	N	N	N	20	B
2	SUNDARAPANDI	47	M	MASON	15 YRS	N	N	N	15	B
3	ABUTHAHIR	52	M	FARMER	25 YRS	1	1	1	25	B
4	SIKKANDAR	48	M	FARMER	17 YRS	1	1	1	17	B
5	BABUJI	45	M	MASON	23 YRS	1	1	1	23	B
6	GURUSAMI	60	M	COMPOUNDER	30 YRS	1	1	1	30	C
7	KUMAR	45	M	MASON	18 YRS	N	N	N	18	C
8	ANGASELVAM	54	M	FARMER	24YRS	1	1	1	24	B
9	ARUMUGAM	52	M	MASON	22YRS	1	1	1	44	C
10	SENTHIL	48	M	CARPENTER	24 YRS	1	1	1	24	C
11	GURUDEV	54	M	FARMER	20 YRS	1	1	1	40	C
12	KUMARAVEL	45	M	BUSINESS	15 YRS	1	1	1	30	C
13	MOHAMMAD	52	M	BUSINESS	26 YRS	1	1	1	26	C
14	RAFIQUE	48	M	EXECUTIVE	15 YRS	1	1	1	15	C
15	SUNDAR	35	M	BUSINESS	10YRS	N	N	N	10	C
16	RAJA	48	M	CONDUCTOR	28 YRS	N	N	N	28	C
17	MURUGAN	37	M	MASON	17 YRS	N	N	N	17	C
18	RAVICHANDRAN	44	M	FARMER	24 YRS	1	1	1	24	B
19	SEETHARAMAN	52	M	DOCTOR	40 YRS	1	1	1	40	C
20	SHELTON	35	M	ENGINEER	10 YRS	N	N	N	10	C
21	ANBALAGAN	38	M	SWEEPER	18 YRS	N	N	N	18	B
22	AYYAPAN	33	M	SWEEPER	18 YRS	N	N	N	36	B
23	SENTHIL KUMAR	37	M	GROCERY	20 YRS	N	N	N	20	C
24	KARTHICK	37	M	GROCERY	18 YRS	N	N	N	18	C
25	MAHENDRAN	52	M	FARMER	28 YRS	2	2	2	42	B
26	RANGAMANI	40	M	BUSINESS	12 YRS	N	N	N	12	C
27	DAVID	34	M	BUSINESS	14 YRS	N	N	N	14	C
28	NAGARAJAN	42	M	MASON	22 YRS	N	N	N	22	B
29	SELVARAJ	32	M	FARMER	10 YRS	N	N	N	10	B
30	RAMSUNDAR	42	M	FARMER	12 YRS	N	N	N	12	C
31	NAGASUBBU	52	M	GROCERY	35 YRS	2	2	2	35	C
32	PONNUSAMY	44	M	MASON	10 YRS	N	N	N	10	C
33	KARUNAS	45	M	SWEEPER	14 YRS	N	N	N	14	B
34	RAHUMAN	52	M	BUSINESS	30 YRS	1	1	1	30	C

35	ILAYARAJA	45	M	FARMER	18YRS	N	N	N	18	C
36	KAMAL	43	M	CARPENTER	18 YRS	N	N	N	18	C
37	RAMAR	37	M	BUSINESS	10 YRS	N	N	N	10	C
38	GURU	55	M	SWEEPER	35 YRS	1	1	1	35	B
39	SUKIRTHA RAJ	58	M	SWEEPER	32 YRS	1	1	1	32	B
40	SHEIK	45	M	SWEEPER	25 YRS	1	1	1	25	B
41	RAJADURAI	56	M	MASON	26 YRS	1	1	1	26	C
42	GIRIDHAR	58	M	BUSINESS	40 YRS	1	1	1	40	C
43	MARTHANDAM	56	M	MASON	42 YRS	1	1	1	42	B
44	RAJAMANI	54	M	CARPENTER	35 YRS	1	1	1	35	C
45	KUPPUSAMY	56	M	BUSINESS	36 YRS	1	1	1	36	C
46	ALBERT	60	M	GROCERY	38 YRS	1	1	1	38	C
47	AROCKIYASAMI	54	M	FARMER	28 YRS	1	1	1	28	B
48	MURUGAN	60	M	FARMER	36 YRS	1	1	1	18	C
49	KANNAN	60	M	CARPENTER	40 YRS	1	1	1	20	C
50	DEVA	52	M	MASON	45 YRS	1	1	1	45	B
N - Normal, 1- Mild, 2- Moderate, B-Bidis, C-Cigarretes										

Normal Spirometry

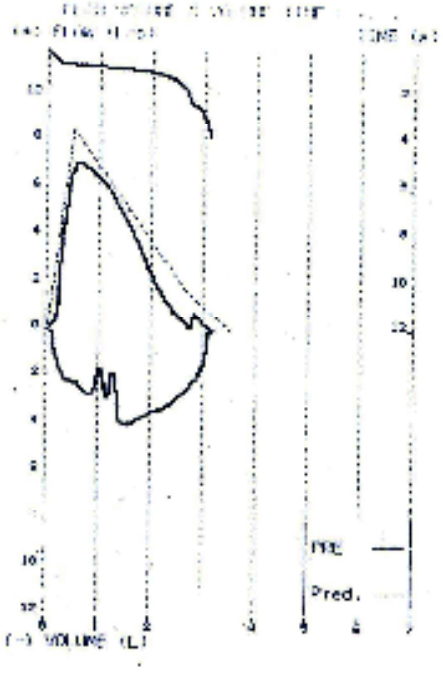


	Pred.		
FVC	3.78	3.73	99
FEV1	3.28	3.18	98
FEV1/FVC	86.7	85.2	103
PEF	8.82	8.02	89
FEF25/75	4.22	3.40	79

FVC	L	3.78	3.54	94
FEV1	L	3.28	3.07	94
FEV1/FVC	%	86.7	86.7	100
FEV6	L	3.78	3.54	94
FEV1/FEV6	%	86.4	86.7	100
PEF	L/s	8.82	7.87	89
FEF25/75	L/s	4.22	3.49	83
FEV3	L	3.58	3.34	93
FEV3/FVC	%	94.6	1000.0	105
FET75%	L/s	4.03	6.51	161
FEF50%	L/s	5.10	3.82	75
FEF75%	L/s	2.20	1.67	76

Normal Spirometry

Mild Obstruction

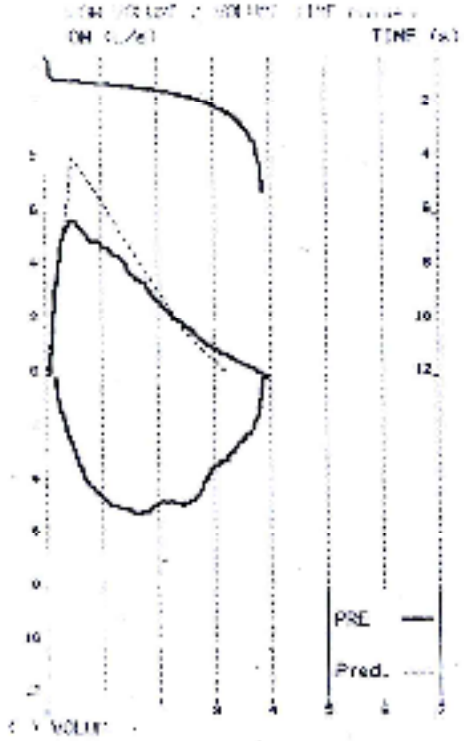


	Pred.		
FVC	3.58	4.15	116
FEV1	2.93	2.59	88
FEV1/FVC	78.1	62.4	79
PEF	8.42	7.11	84
FEF25/75	4.01	1.61	40

FVC	L	3.58	4.13	115
FEV1	L	2.93	2.59	88
FEV1/FVC	%	78.1	62.7	80
FEV6	L	3.58	4.13	115
FEV1/FEV6	%	78.1	62.7	80
PEF	L/s	8.42	7.11	84
FEF25/75	L/s	4.01	1.61	40
FEV3	L	3.38	3.11	92
FEV3/FVC	%	94.3	75.4	80
FET75%	L/s	4.04	6.79	168
FEF50%	L/s	4.58	4.54	99
FEF75%	L/s	2.15	1.67	78

Mild Obstruction

Normal Spirometry



FVC	L	3.92	3.89	117
FEV1	L	2.76	2.80	108
FEV1/FVC	%	70.1	76.9	92
FEV6	L	3.92	3.89	117
FEV1/FEV6	%	80.0	78.9	93
PEF	L/s	8.18	5.80	71
FEF25/75	L/s	3.21	2.60	68
FEF75/85	L/s	1.72	1.16	69

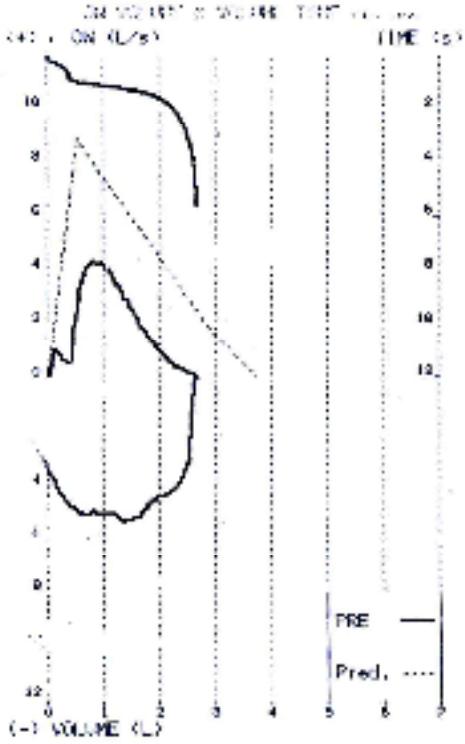
SPIROMETRY INTERPRETATION:
Normal Spirometry

QUALITY CONTROL GRADE: D

REPEATABILITY: None

Note by spirolab III Ver 2.0 (N-00017)

Moderate Obstruction



FVC	L	3.81	2.66	96
FEV1	L	3.19	2.19	69
FEV1/FVC	%	80.6	82.3	102
FEV6	L	3.81	2.66	70
FEV1/FEV6	%	83.6	82.3	98
PEF	L/s	8.87	4.91	49
FEF25/75	L/s	4.07	2.47	61
FEF75/85	L/s	2.03	.96	48

SPIROMETRY INTERPRETATION:
Possible Moderate Obstruction

QUALITY CONTROL GRADE: F

REPEAT Test and Short Factor

REPEATABILITY: None

Note by spirolab III Ver 2.0 SN-00017

Anatomy of Lungs